

Maternal Obesity Affects Newborn Somatometrics and Vital Parameters in a Gender Typical Manner – Evidence for the Male Disadvantage Hypothesis?

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ABSTRACT

According to the male disadvantage hypothesis male fetuses react more sensitive to maternal stress factors during gestation. In the present study the gender typical impact of maternal prepregnancy overweight and obesity as well as gestational weight gain on newborn somatometrics was tested on basis of births records of 7565 births, which took place in Vienna Austria. Maternal weight status was determined at the beginning of pregnancy according to the WHO recommendations. Newborns were measured immediately after birth. With increasing maternal prepregnancy weight status and increasing gestational weight gain birth weight, birth length and head circumference increased too. Among male newborns however the increase was lower than among female ones. With increasing maternal weight status the prevalence of macrosome newborns increased significantly. Among girls however this increase was significantly higher. Male fetuses seem to react more sensitive to a higher maternal weight status in comparison to girls. These results can be interpreted in sense of the so called male disadvantage hypothesis.

Key words: Maternal obesity, pregnancy outcome, birth weight, male disadvantage hypothesis

Introduction

Obesity is increasing worldwide at an alarming rate and represents the fastest growing health problem in developed countries¹. Today more than 1.1 billion adults worldwide are classified as overweight, 312 million of them as obese². This tendency is a matter of concern because obesity results not only in social stigmatisation and deleterious social and economic consequences³, obesity increases also risks for many morbid conditions such as hypertension, diabetes mellitus dyslipidemia, coronary disease and some cancers such as breast cancer or colon cancer^{4,5}.

A special problem represents obesity among women of childbearing age^{6,7}. In this age group obesity rates have increased dramatically during the last twenty years^{8,9} with marked consequences for female reproduction. Existing research supports a link between excessive obesity and conditions which may have adverse effects on the probability of successful conception⁷. Obese women may suffer from PCOS, insulin resistance, hyperinsulinaemia, hyperandrogenemia, increased peripheral aromatization

from androgens to estrogens, altered gonadotropin secretion, decreased SHBG levels, increased leptin levels and altered neuroregulation of the hypothalamic-pituitary-gonadal axis¹⁰. In this case female obesity is related positively with infertility or subfertility. Furthermore obesity is associated with unfavourable IVF/ICSI cycle outcomes as evidenced by lower pregnancy rates¹¹. But even if conception takes place maternal obesity is known to be a risk factor of several serious complications such as increased maternal morbidity, including gestational diabetes, infectious morbidity, preclampsia postpartum haemorrhage or delivery of large for date or macrosome babies^{7,12,13}. Additional several studies reported an increased rate of caesarean deliveries among obese mothers^{14–16}. Some studies have also shown an elevated risk for heart defects but also neurotubal effects such as spina bifida¹⁷. Furthermore long term effects of maternal obesity have been described: maternal obesity at conception leads to fetal programming of offspring, which could result in obesity of the offspring in later life¹⁸. From these

observations mentioned above we can conclude, that prepregnancy obesity represents an important stress factor for mother and child. Since more than 40 years it is well known that prenatal stress affects male and female fetuses in a different manner¹⁹. Male fetuses react more sensitive to stress factors, resulting in a higher male prevalence among abortions and stillbirths²⁰. These observations resulted in the formulation of the so called »male disadvantage hypothesis«^{21–23}. As mentioned above prepregnancy obesity is mentioned as a stress factor for mother and child. But do male and female fetuses react different on maternal prepregnancy obesity? In the present paper the effects of maternal weight status, especially the effects of overweight and obesity on newborn somatometric characteristics are analysed, with respect to gender differences.

Material and Methods

Data set

The present study is based on a data set of 22189 singleton births which took place at the University Hospital for Gynaecology and Obstetrics in Vienna, Austria between 1985 and 2000. The Viennese University Hospital for Gynaecology and Obstetrics is one of the largest births clinics in Vienna. During the early seventies Austria had developed a highly sophisticated system of pre- and postnatal care, which included seven check-ups during pregnancy starting at the 8th week of gestation, and eight postnatal check ups of the child until the fourth year of life, free of charge. This system helped to reduce the neonatal and child mortality rate in Austria dramatically²⁴. Prenatal check ups were performed in the consulting rooms of gynaecologists or at the clinic where birth was scheduled to take place. All data were documented at the hospital and in the so called »mother-child-passport« which belongs to the mother. During the prenatal check ups, maternal somatometric factors are also documented as well as characteristics of the foetus. In our study we included the data of 7565 non smoking primiparae women ageing between 19 and 39 years ($\bar{X}=26.1\pm 5.2$ yrs) at the time of giving first birth. The following inclusion criteria were used: term births (between 38 and 41 week of gestation), all prenatal check ups of the mother-child passport were completed, the delivery of single infants without congenital malformations, no registered maternal diseases before and during pregnancy, no hypertension (BP < 150/90 mmHg), no protein or glucose in the urine, no pregnancy related immunisation. Furthermore coincident medical diseases such as diabetes mellitus or nephropathy, nicotine consumption, drug or alcohol abuse and twin birth or IVF were strict exclusion criterions. Gestational age was calculated in terms of the number of weeks from the beginning of the last menstrual bleeding to the date of delivery (=duration of amenorrhoea) and by two consecutive ultrasound examinations performed before the 12th week of gestation. All probands belonged predominantly to the urban middle class. All women had finished school, or were at school (espe-

cially the adolescent mothers in the sample) at the time of pregnancy. The study was carried out in compliance with »Ethical principles for medical research involving human subjects« of Helsinki Declaration and approved by bioethics committee. The study is only a small part of a large project concerning pregnancy outcome in association with maternal biomedical parameters. All mothers who had given birth during the study period (1985–2000) were informed about the objectives of the study and had the right to withdraw.

Maternal characteristics

Beside the documentation of menarcheal age, and chronological age at the time of delivery, the gynaecological age was calculated (Chronological age minus menarcheal age). Furthermore the following somatometric data of all women were documented:

Stature, prepregnancy weight (PPW), weight at the end of pregnancy (EPW), weight gain during pregnancy (PWG), and the pelvic measures *Distantia spinarum* (DSP) and *Distantia christarum* (DCR)^{25,26}. Prepregnancy weight was estimated by means of the retrospective method and the first weight determination, which was carried out at the first prenatal visit (8th week of gestation). In order to determine prepregnancy weight the mean value of the retrospective estimated weight and the weight at the 8th week of gestation was calculated.

Maternal weight status

Weight status was estimated by means of Body mass index (kg/cm²). Weight status was classified according to the BMI categories recommended for adults by the WHO²⁷:

- BMI < 16.00: severe underweight
- BMI 16.00–16.99: underweight
- BMI 17.00–18.49: slight underweight
- BMI 18.50–24.99: normal weight
- BMI 25.00–29.99: overweight
- BMI 30.00–39.99: obese
- BMI > 40.00 morbid obese

Newborn characteristics

One and five minute APGAR Scores^{28,29} for evaluation of the newborn were determined and all newborns were measured immediately after birth. The following parameters were taken directly from the newborn²⁶:

Birth weight, birth length, head circumference, Diameter frontooccipitalis. Birth weight status was defined according to the recommendations of the WHO³⁰: a low birth weight as defined as < 2500 g, a normal birth weight as 2500–4000 g and a macrosomia as a birth weight > 4000 g.

Statistical analysis

Statistical analyses were performed by means of SPSS for Windows Program Version 16.0 (Microsoft corp.). After calculation of descriptive statistics (\bar{X} , SDs) group differences were tested regarding their statistical signifi-

cance using student t-tests, ANOVAS and Duncan post hoc tests. Crosstabs (χ^2) were calculated to test frequency differences with respect to their statistical significance. Furthermore linear regression analyses were calculated to test the association between maternal weight status as well as gestational weight gain and newborn size.

Results

Maternal and newborn characteristics

Maternal and newborn characteristics are listed in Tables 1 and 2. More than 80% of the mothers were classified as normal-weight before pregnancy, less than 15% of the mothers were classified as overweight, 3.6% as obese. Only 0.3% of the mothers corresponded to the definitions of morbid obesity. The majority of women (45.4%)

TABLE 1
PRESENTATION OF MATERNAL CHARACTERISTICS (X, SD, %)

Maternal characteristics	% \bar{X} (SD)
Age (yr)	26.1 (5.2)
Stature height (cm)	163.3 (6.5)
Prepregnancy weight (PPW)	59.8 (10.3)
Prepregnancy weight status (BMI)	22.36 (3.61)
Normal weight BMI 18.50–24.99	81.8%
Overweight BMI 25.00–29.99	14.4%
Obese BMI 30.00–30.99	3.6%
Morbid obese >40.00	0.3%
End of pregnancy weight (EPW)	73.4 (12.3)
Pregnancy weight gain	12.9 (5.5)
<10 kg	25.1%
10–15 kg	45.4%
>15 kg	29.5%

TABLE 2
PRESENTATION OF NEWBORN CHARACTERISTICS ACCORDING TO NEWBORN SEX (X, SD, p, Student t-tests)

	female \bar{X} (SD)	male \bar{X} (SD)	Sig (p)
Birth weight	3297.5 (425.4)	3441.4 (430.1)	<0.001
Birth length	49.4 (1.9)	50.3 (1.8)	<0.001
Head circumference	34.1 (1.3)	34.6 (1.4)	<0.001
Diameter frontooccipitalis	11.2 (0.7)	11.4 (0.8)	<0.001
Apgar score 1 minute	8.7 (1.2)	8.6 (1.3)	<0.007
Apgar score 5 minutes	9.8 (0.7)	9.7 (0.7)	<0.007
Weight status			
Low birth weight <2500 g	2.4%	1.2%	<0.001
Normal weight 2500–4000 g	92.1%	88.1%	
Macrosomia >4000 g	5.5%	10.7%	

gained between 10 and 15 kg during pregnancy. About 25% gained less than 10 kg, while nearly 30% experienced a weight gain of more than 15 kg. Female and male newborns differed significantly in birth weight, birth length, head circumference, diameter frontooccipitalis and in the Apgar scores 1 and 5 minutes after birth. While boys always surpassed the girls in somatometric parameters, girls showed higher APGAR score values. Newborn girls and boys differed also significantly regarding weight status. Low birth weight (<2500 g) was significantly more often found among newborn girls, while macrosomia (>4000 g) was significantly more frequent among newborn boys (Tables 1 and 2).

Gestational weight gain and new born characteristics

With increasing pregnancy weight gain birth weight, birth length and head circumference increased significantly. This was true of both genders, however among girls birth weight increased significantly higher than among boys ($p < 0.01$). Among girls the difference between the lowest mean birth weight and the highest mean birth weight was 236.6 g, among boys birth increased only 205.2 g. A significantly higher benefit of increased gestational weight gain among girls was also found for birth length and head circumference (Table 3). Gender differences in birth weight, birth length and head circumference decrease with increasing pregnancy weight gain (Table 3). As to be seen in Table 4 gestational weight gain was significantly positively related with newborn somatometric characteristics. With increasing weight gain newborns are heavier, longer and had a higher head circumference. This was true of both genders. The APGAR scores did neither differ significantly between the two genders, nor increase with increasing weight gain. In contrast APGAR scores decreased with increasing gestational weight gain in both genders.

Maternal prepregnancy weight status and newborn characteristics

With increasing prepregnancy weight status birth weight, birth length and head circumference increased significantly. This was true of girls as well as of boys. The benefit of higher prepregnancy weight status for newborn somatometrics however was significantly higher among girls. The difference between mothers of the lowest prepregnancy weight status category and those of the highest prepregnancy weight of status category was 252.5 g among girls, but only 192.1 g among boys. A similar trend was observed for birth length. Regarding head circumference gender differences increase with increasing prepregnancy weight status (Table 3). As to be seen in Table 4 prepregnancy weight status was significantly positively related with newborn somatometric characteristics. This was true of both genders. Concerning the APGAR score no marked differences between newborn girls and boys could be observed. With increasing prepregnancy weight however, the APGAR scores of boys and girls decreased.

TABLE 3
GENDER DIFFERENCES IN NEWBORN FEATURES ACCORDING TO MATERNAL PREPREGNANCY WEIGHT STATUS AND PREGNANCY WEIGHT GAIN

	Birth weight (in g)			Birth length (in cm)			Head circumference (in cm)			APGAR 1			APGAR 5		
	Female	Male	Diff	Female	Male	Diff	Female	Male	Diff	Female	Male	Diff	Female	Male	Diff
	\bar{X} (SD)	\bar{X} (SD)		\bar{X} (SD)	\bar{X} (SD)		\bar{X} (SD)	\bar{X} (SD)		\bar{X} (SD)	\bar{X} (SD)		\bar{X} (SD)	\bar{X} (SD)	
Pregnancy weight gain (PWG)															
<10 kg	3195.3 (413.0)	3347.2 (433.1)	151.9	48.9 (1.9)	49.9 (1.9)	1.1	33.9 (1.4)	34.5 (1.4)	0.6	8.8 (1.1)	8.7 (1.2)	-0.1	9.8 (0.6)	9.8 (0.6)	-0.05
10–15kg	3314.5 (422.6)	3443.8 (414.3)	129.3	49.4 (1.9)	50.3 (1.8)	0.9	34.1 (1.3)	34.6 (1.4)	0.5	8.6 (1.2)	8.6 (1.3)	-0.01	9.7 (0.7)	9.7 (0.9)	-0.01
>15kg	3431.9 (427.7)	3552.4 (440.3)	120.4	49.8 (1.7)	50.7 (1.7)	0.9	34.4 (1.4)	34.8 (1.4)	0.4	8.6 (1.2)	8.5 (1.3)	-0.15	9.7 (0.7)	9.6 (0.8)	-0.08
Sig (p)	<0.001			<0.01			n.s.			n.s.			n.s.		
Maternal prepregnancy weight status (BMI)															
18.50–24.99	3277.5 (414.0)	3424.6 (420.6)	147.1	49.3 (1.9)	50.3 (1.9)	0.9	34.1 (1.3)	34.6 (1.4)	0.5	8.7 (1.2)	8.6 (1.2)	-0.05	9.8 (0.7)	9.8 (0.7)	-0.01
25.00–29.99	3395.2 (445.9)	3547.8 (447.1)	152.5	49.6 (1.0)	50.6 (1.8)	0.9	34.3 (1.4)	34.9 (1.4)	0.6	8.6 (1.4)	8.6 (1.4)	-0.05	9.7 (0.9)	9.7 (0.7)	-0.02
30–39.99	3491.7 (420.6)	3598.6 (467.4)	106.9	49.9 (1.8)	50.9 (1.8)	1.0	34.4 (1.3)	35.0 (1.4)	0.6	8.6 (1.3)	8.3 (1.9)	-0.27	9.8 (0.5)	9.6 (1.4)	-0.22
>40.00	3530.0 (692.3)	3516.7 (422.3)	13.3	50.1 (1.7)	50.4 (0.9)	0.3	34.8 (1.7)	35.6 (1.5)	0.8	0.87 (0.9)	8.0 (1.5)	-0.67	9.8 (0.5)	9.5 (0.7)	-0.25
Sig (p)	<0.01			<0.01			<0.05			n.s.			n.s.		

TABLE 4
IMPACT OF GESTATIONAL WEIGHT GAIN, PREPREGNANCY WEIGHT AND PREPREGNANCY WEIGHT STATUS ON NEWBORN SOMATOMETRICS, MULTIPLE REGRESSION ANALYSES

Dependent variable: Birthweight	R ²	Coefficient	Sig	95% confidence interval
Female				
Prepregnancy weight status	0.10	25.64	0.000	20.84–30.44
Weight gain		19.46	0.000	16.01–22.92
Male				
Prepregnancy weight status	0.10	26.44	0.000	21.65–31.27
Weight gain		17.21	0.000	13.86–20.62
Dependent variable: Birth length	R ²	Coefficient	Sig	95% confidence interval
Female				
Prepregnancy weight status	0.05	0.08	0.000	0.06–0.09
Weight gain		0.06	0.000	0.05–0.08
Male				
Prepregnancy weight status	0.06	0.09	0.000	0.07–0.11
Weight gain		0.06	0.000	0.04–0.07
Dependent variable: head circumference	R ²	Coefficient	Sig	95% confidence interval
Female				
Prepregnancy weight status	0.04	0.05	0.000	0.04–0.07
Weight gain		0.04	0.000	0.03–0.05
Male				
Prepregnancy weight status	0.05	0.06	0.000	0.05–0.08
Weight gain		0.02	0.000	0.01–0.04

Prevalence of macrosome and low weight newborn

As to be seen in Figures 1 and 2 the prevalence of macrosomia (>4000 g) increased significantly ($p < 0.000$) with increasing gestational weight as well as with increasing prepregnancy weight status. This was true of both genders however the prevalence of macrosomia increased relatively more among girls. The prevalence of low weight newborns (<2500 g) however decreased significantly ($p < 0.000$) with increasing gestational weight gain as well as maternal prepregnancy weight status. This was also true of both genders (Figures 3 and 4).

Discussion

More than 40 years ago the so called »male disadvantage hypothesis« was introduced by Richard Naeye in order to explain the increased risk of perinatal morbidity and morbidity in boys in comparison with girls¹⁹. Today the newborn male disadvantage is a well established fact. There is not only a 30% predominance of male fetuses among early spontaneous, chromosomally normal abortions described²⁰, boys have also a slightly increased risk to be affected by congenital malformations, show a higher mortality and more postnatal complications as a result of low birth weight, show more often depressed Apgar scores and had a higher frequency of respiratory distress syndrome or lung related injuries and disabilities^{31,32}. In comparison to girls, boys are more often born prematurely and were generally less stable after birth mainly due to pulmonary morbidity but also due to intracranial haemorrhage and urinary tract infections^{22,33}. But also after prenatal and perinatal period the increased risks for boys seem to continue. Sudden infant death syndrome, for instance, is more often found among boys than among girls, and during childhood boys suffer more often from respiratory disease, asthma, gastroenteritis, behaviour disorders, intellectual disability and accidents than girls³¹. Although the male disadvantage hypothesis was proved by many different studies²², little is known about possible mechanisms contributing to the increased morbidity and mortality in newborn boys. Mainly physiological differences in cerebral blood flow, neonatal stress², gender related differences in CSG Levels of IL-8 and antioxidants³⁴ and gender differences in leptin levels³⁵ have been discussed as reasons for increased morbidity and mortality in boys. Male fetuses seem to be more vulnerable to an adverse intrauterine environment than female fetuses. Although male newborns are more vulnerable, several studies indicate that male newborns are heavier and longer and exhibit larger head circumferences than their female counterparts^{36–38}. According to the male disadvantage hypothesis however we may hypothesize that maternal stress factors during pregnancy may have an increased adverse effect on fetal growth in males in comparison to females. In the present study we focused on maternal obesity as an important stress factor. As pointed out in the introduction section maternal obesity is associated with an adverse pregnancy outcome such as increased maternal morbidity, including gesta-

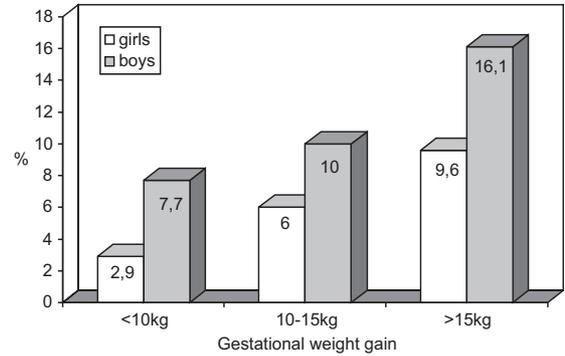


Fig.1. Prevalence of macrosome newborns according to gestational weight gain.

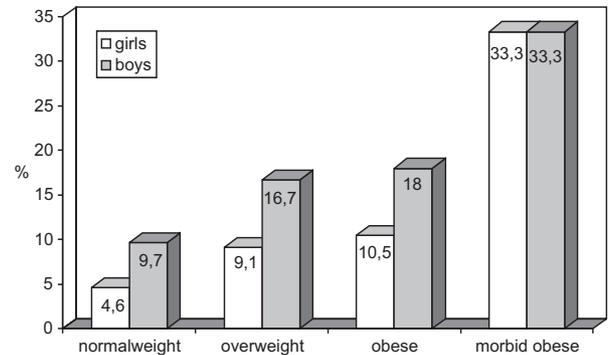


Fig. 2. Prevalence of macrosome newborns according to maternal prepregnancy weight status.

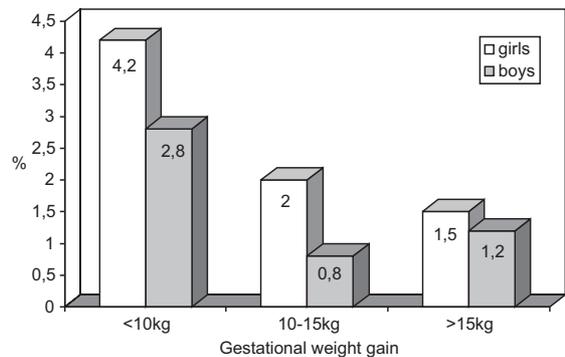


Fig. 3. Prevalence of low weight newborns according to gestational weight gain.

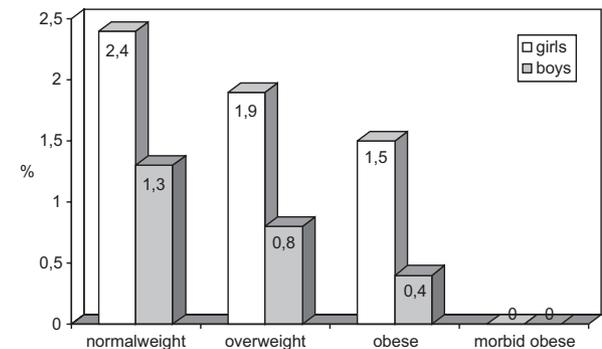


Fig. 4. Prevalence of low weight newborns according to maternal prepregnancy weight status.

tional diabetes, infectious morbidity, preclampsia postpartum haemorrhage or delivery of large for date or macrosome babies^{6,7,12,13,17}. We hypothesized that maternal obesity before and during gestation reduce somatometric gender differences in newborns. The results of the present study corroborate this idea. Although birthweight, birth length and head circumference increased with increasing maternal weight status and gestational weight gain, the surplus among male newborns is significantly lower than that of newborn girls. Another observation corroborates this result. With increasing gestational weight gain as well as with increasing maternal prepregnancy weight status the prevalence of macrosome newborns increase significantly. Although this was true of both genders, the increase among girls was significantly higher. While for instance the prevalence of macrosomia between the group with the lowest gestational weight gain and the group with the highest gestational weight gain doubled among boys it trebled among girls. Concerning prepregnancy weight status the prevalence of macrosome girls was 8 times higher among morbid obese mothers in comparison to normal-weight mothers. Among boys in contrast only a 3 times higher prevalence among morbid obese mothers could be observed.

These results plead for a gender typical effect of maternal weight status and gestational weight gain on newborn somatometrics because the stress factor maternal obesity seems to have a higher impact on male than on female foetuses. There is no doubt that newborn girls and boys differ significantly in somatometric dimensions^{31,36–38}. These gender differences in newborn dimensions are not only found among human newborns, non-human primates exhibit also gender differences in newborn somatometrics^{39,40}. In general newborn boys are

heavier and longer, and exhibit a higher head circumference than newborn girls, although girls in contrast exhibit a higher amount of subcutaneous fat distribution⁴¹. Especially birth weight was affected by maternal stress factors.

The effects of maternal prepregnancy weight status and pregnancy weight gain on newborn size in general are well known⁴², the gender typical differences of the impact of maternal weight status and gestational weight gain on newborn somatometrics however, corroborates the male disadvantage hypothesis. From a physiological point of view these results may be explained by a sex-biased environmental sensitivity caused by gender typical differences in leptin levels and general hormonal function⁴³. Boys grow faster and have a higher metabolic rate than girls during gestation, however when oxygen is limited they might deplete available resources more rapidly⁴⁴. From an evolutionary point of view gender differences in early vulnerability may be attributed to the natural selection of optimal maternal strategies to maximize life time reproductive success^{45–48}. Wells tried to explain the increased male sensitivity to adverse environmental conditions by means of the Trivers-Willard hypothesis of differential parental investment⁴⁹. Trivers and Willard assumed that in vertebrates nearly all females mate successfully, while male reproductive success is only ensured under favourable environmental conditions. Therefore parents, especially mothers, manipulate the sex ratio of their offspring dependent on environmental conditions. Natural selection is therefore predicted to favour increased male vulnerability to general factors such as infectious diseases or malnutrition because of its role on optimizing maternal reproductive fitness^{45,50}.

REFERENCES

- GINTER E, SIMKO V, Bratisl Lek Listy, 109 (2008) 224. — 2. WHO, Global strategy on diet, physical activity and Health (WHO, Geneva, 2009). — 3. LATNER JD, STUNKARD AJ, *Obes Res*, 11 (2003) 452. — 4. GEISS HC, PARHOFER KG, SCHWANDT P, *Int J Obes*, 25 (2001) 830. — 5. MCGEE DL, *Ann Epidemiol*, 15 (2005) 87. — 6. CROGSWELL ME, PERRY GS, SCHIEVE LA, DIETZ WH, *Prim Care Update Ob/Gyn*, 8 (2001) 89. — 7. PANDEY S, BHATTACHARYA S, *Womens Health*, 6 (2010) 107. — 8. VAHRATIAN A, *Matern Child Health J*, 13 (2009) 268. — 9. FRISCHKNECHT F, BRÜHWILER H, RIAO L, LÜSCHER KP, *Swiss Med Wkly*, 139 (200) 52. — 10. PARIHAR M, *Rev Gynecol Pract*, 3 (2003) 120. — 11. AWARTANI KD, NAHAS S, AL HASSAN SH, DEERY MA, COSKUN S, *Reprod Biol Endocrinol*, 7 (2009) 52. — 12. BAETEN JM, BUSKUSI EA, LAMBE M, *Am J Pub Health*, 91 (2001) 436. — 13. KERRIGAN AM, KINGDON C, *Midwifery*, 26 (2010) 138. — 14. BROST BC, GOLDENBERG R, MERCER BM, IAMS JD, MEIS PJ, MOAWARD AH, NEWMAN RB, MIODOVNIK M, CARITIS SN, THURNAU GR, BOTTOMS SF, DAS A, MCNELLIS D, *Am J Obstet Gynecol* 177 (1997) 333. — 15. YOUNG TK, WOODMANSEE B, *Am J Obstet Gynecol*, 187 (2002) 312. — 16. WEISS JL, MALONE FD, EMIG D, BALL RH, NYBERG DA, COMSTOCK CH, SAADE G, EDDLEMAN K, CARTER S, CRAIGO SD, CARR SR, DALTON ME, *Am J Obstet Gynecol*, 190 (2004) 1091. — 17. WATKINS ML, RASMUSSEN SA, HONEIN MA, BOTTO LD, MOORE CA, *Pediatrics*, 111 (2003) 1152. — 18. SHANKAR K, HARELL A, LUI X, GILCHRIST JM, RONIS MJJ, BADGER TM, *Am J Physiol Regul Integr Comp Physiol*, 294 (2008) 528. — 19. NAEYE RL, BURT LS, WRIGHT DL, BLANC WA, TATTER D, *Pediatrics*, 48 (1971) 902. — 20. HASSOLD T, QUILLEN SD, YAMANE JA, *Ann Hum Genet*, 47 (1983) 39. — 21. CAGNACCI A, RENZI A, ARANGINO S, ALESSANDRINI C, VOLPE A, *Hum Rep*, 18 (2003) 885. — 22. STEVENSON DK, VERTER J, FANAROFF AA, OH W, EHRENKRANZ RA, SHANKARAN S, DONOVAN EF, WRIGHT LL, LEMONS JA, TYSON JE, KORONES SB, BAUER CR, STOLL BJ, *Arch Dis Child Fetal Neonatal Ed*, 83(2000) 182. — 23. STEVENSON DK, TYSON JE. Beware of the weaker sex: don't get too close to your twin brother, *Pediatrics*, 120 (2007) 638. — 24. WALDHOER T, HAIDINGER G, LANGASSER J, TUOMILEHTO J, *Wkly Klin Wschr*, 108 (1996) 643. — 25. KNUSSMANN R, *Somatometrie*. In: KNUSSMANN R (Ed) *Anthropologie* (Fischer Verlag, Stuttgart, 1988). — 26. PSCHYREMBEL W, *Handbuch der gynäkologischen Praxis*. (Springer Verlag, Berlin, 1976). — 27. WHO. *Physical status: the use and interpretation of anthropometry*. (WHO Technical Report Series 854, Geneva, 1997). — 28. JONETT RJ, WARFORD HS, KREINICK C, WATERKOTTE GW, *Am J Obstet Gynecol*, 140 (1981) 206. — 29. FORSLAD K, KÄLLEN K, MARSAL K, HELLSTRÖM-WESTAS L, *Acta Paediatrica*, 96 (2007) 166. — 30. WHO, *World Health Status Quarterly*, 33 (1980) 197. — 31. ELSMEN E, STEEN M, HELLSTRÖM-WESTAS L, *JMHG*, 4 (2004) 303. — 32. FINNSTRÖM O, *Acta Paediatrica*, 93 (2004) 1154. — 33. THOMAS MR, MARSTON L, RAFERTY GF, CALVERT S, MARLOW N, PEACOCK JL, GRENOUGH A, *Arch Dis Child Fetal Neonatal Ed*, 91 (2006) 197. — 34. HUSSEIN MH, DAOUD GA, KAKITA H, HATTORI A, MURAI H, YASUDA M, MIZUNO K, GOTO K, OZAKI Y, ITO T, TANAKA T, FUKUDA S, KATO I, FUJIMOTO S, SUZUKI S, SOBAJIMA H, TOGARI H, SHOCK, 28 (2007) 154. — 35. PARDO IMCG, GELONEZE B, TAMBASCIA MA, PEREIRA JL, BARROS FILHO AA, *J Pediatr*, 80 (2004) 305. — 36. CRAWFORD MA, DOYLE W, MEADOWS N, *Hum Re-*

- prod, 2 (1987) 517. — 37. MARSAL K, PERRSON PH, LARSEN T, LILJA H, SELBING A, SULTAN B, Acta Paediatr, 85 (1996) 843. — 38. YANKOVA I, Rev Environ Health, 20 (2005) 65. — 39. JOFFE TH, TARANTAL AF, RICE K, LELAND M, OERKE AK, RODECK C, GEARY M, HINDMARSH P, WELLS JCK, AIELLO LC, Am J Phys Anthropol, 126 (2005) 97. — 40. GEARY MPP, PRINGLE PJ, RODECK CH, KINGDOM JCP, HINDMARSH PC, J Clin Endocrinol Metab, 88 (2003) 3708. — 41. RODRIGUEZ G, SAMPER MP, VENTURA P, MORENO LA, OLIVARES JL, PEREZ-GONZALEZ JM, Eur J Pediatr, 163 (2004) 457. — 42. KIRCHENGAST S, HARTMANN B, Ann Hum Biol, 25 (1998) 17. — 43. MURPHY VE, SMITH R, GILES WB, CLIFTON VL, Endocrine Reviews, 27 (2006) 141. — 44. BENNET L, BOOTH LC, AHMED-NASEF N, DEAN JM, DAVIDSON J, QUAEDACKERS JS, GUNN AJ, Am J Physiol Regul Integr Comp Physiol, 293 (2007) 1280. — 45. WELLS JCK, J Theor Biol, 202 (2000) 65. — 46. LUMMAA V, JOKELA J, HAUKIOJA E, J Anim Ecol, 70 (2001) 739. — 47. LUMMAA V, CLUTTON-BROCK T, Trends Ecol Evol, 17 (2002) 141. — 48. BEISE J, VOLAND E, History Family, 7 (2002) 515. — 49. TRIVERS RL, WILLARD DE, Science, 179 (1973) 90. — 50. KIRCHENGAST S, HARTMANN B, J Life Sci, 1 (2009) 63.

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UTJECAJ MAJČINE PRETILOSTI NA SOMATOMETRIJU NOVOROĐENČADI I VITALNIH PARAMETARA U ODNOSU NA SPOL DIJETETA – DOKAZ ZA HIPOTEZU MUŠKOG NEPOVOLJNOG POLOŽAJA?

SAŽETAK

Prema hipotezi muškog nepovoljnog položaja, muški fetusi reagiraju osjetljivije na majčine stresne čimbenike tijekom trudnoće. U ovom istraživanju utjecaj majčine predtrudničke prekomjerne težine i pretilost, kao i gestacijska težina na somatometriju novorođenčadi, testiran je na temelju evidencije 7565 rođenih, u Beču, Austrija. Status majčine težine određen je na početku trudnoće prema preporukama SZO. Novorođenčad je izmjerena odmah nakon rođenja. Uz povećanje statusa pretrudničke težine majke i povećanje gestacijske težine, povećana je i porođajna težina, porođajna dužina i opseg glave. Među muškom novorođenčadi, porast je bio manji nego kod one ženske. S povećanjem statusa majčine težine, prevalencija veće novorođenčadi značajno je porasla. No, među ženskom novorođenčadi to povećanje je znatno veće. Muški fetusi čini se da reagiraju osjetljivije na viši status majčine težine u odnosu na ženske fetuse. Ovi rezultati se mogu tumačiti u smislu tako zvane hipoteze muškog nepovoljnog položaja.