

METABOLIC SYNDROME AND BIRTH ANTHROPOMETRIC DATA IN PRADER-WILLI SYNDROME.



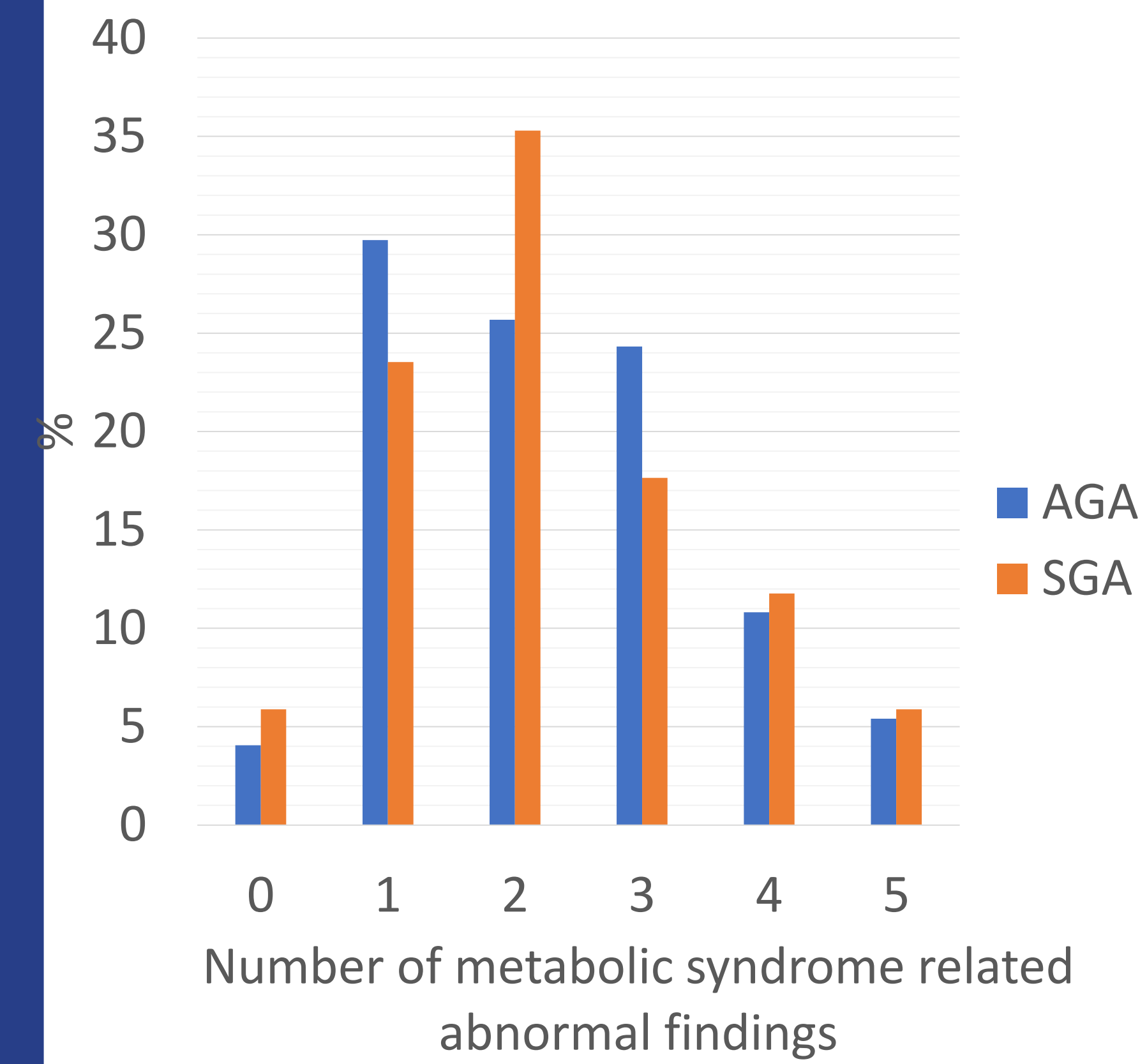
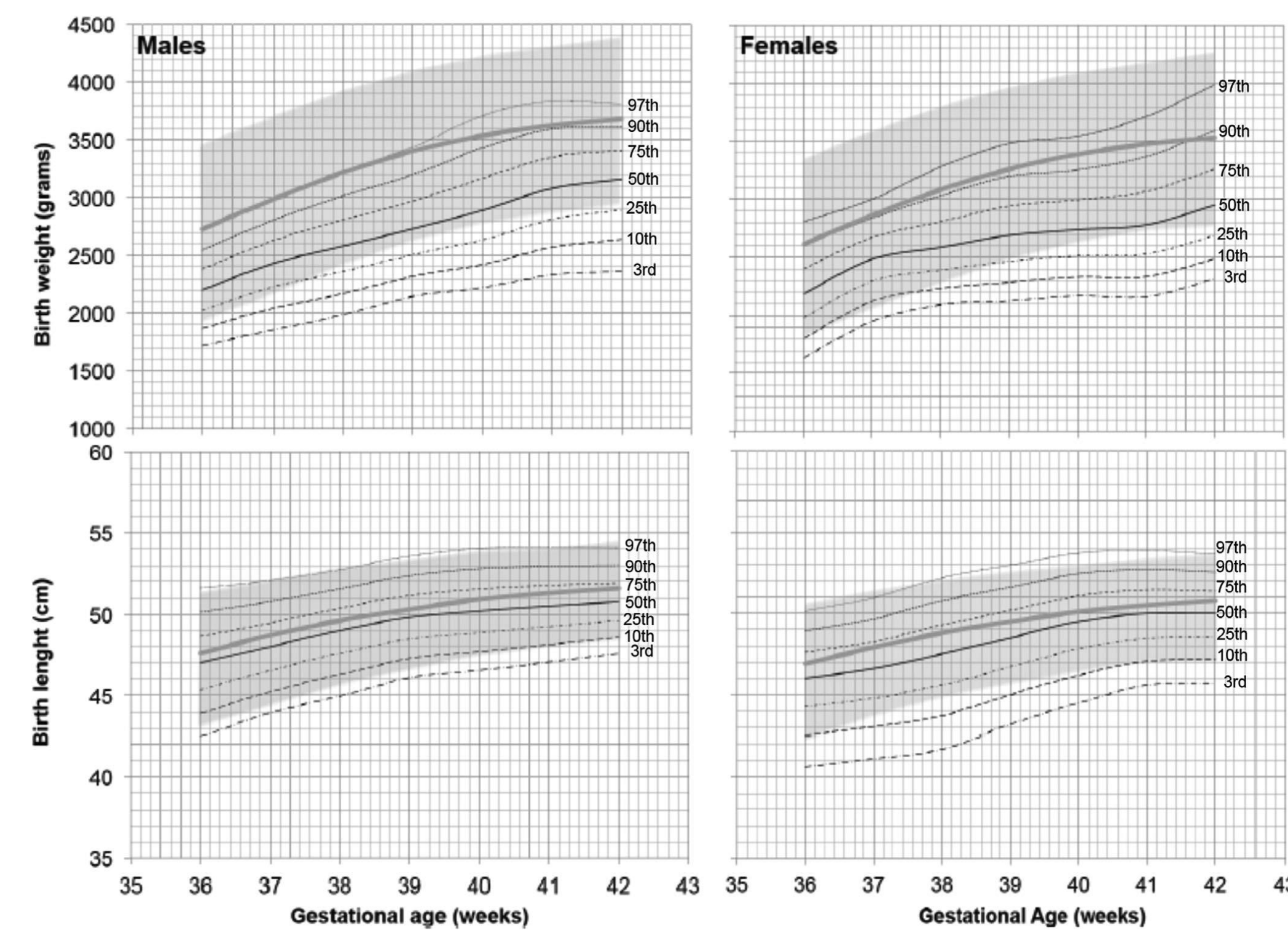
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INTRODUCTION

Previous studies showed that non-obese children and adults with Prader-Willi syndrome (PWS) have a low frequency of metabolic syndrome (MetS), while obese ones have a frequency similar to that of non-PWS obese. It is known that individuals born small for gestational age (SGA) have a greater predisposition to the development of MetS. Recent neonatal percentiles of subjects with PWS (Salvatoni et al, Am J Med Genet Part A, 2019) documented a defect in weight of half kg and in length of 1 cm compared to general population. Moreover, females with a 15q11-13 deletion (DEL15) resulted shorter than those with maternal uniparental disomy of chromosome 15 (mUPD15).



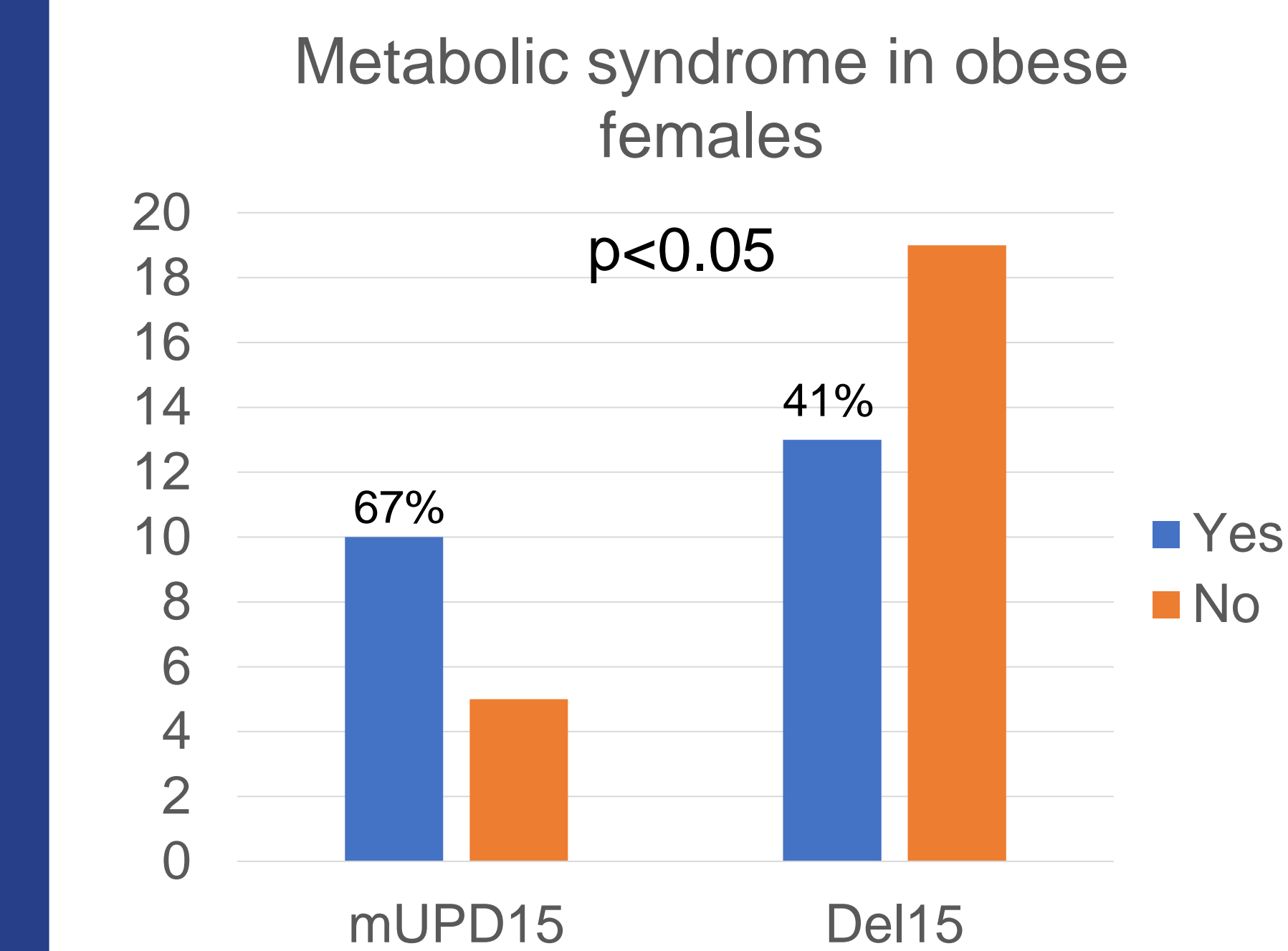
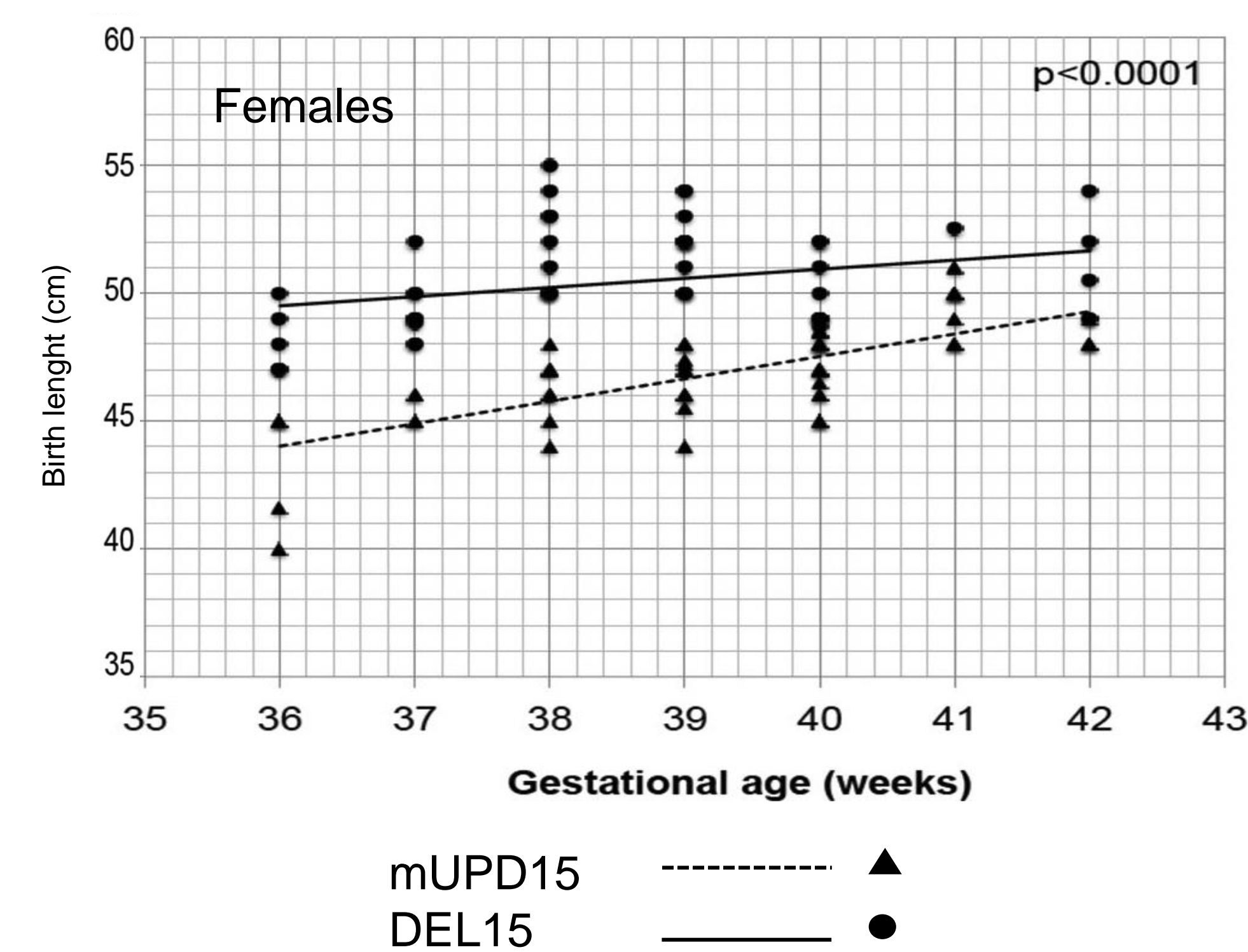
	Total	AGA	SGA	p
DEL15/UPD15	69/22	57/17	12/5	>0.05
Gender (F/M)	59/32	44/30	15/2	<0.05
GA (weeks)	40 (1)	40 (2)	40 (1)	>0.05
BW (g)	2750 (687)	2855 (450)	2250 (223)	<0.0001
SDS-BW	0.11 (1.11)	0.18 (0.716)	2.84 (73.653)	<0.0001
Age (yrs)	27.6 (5.3)	27.7 (4.6)	24.2 (7.8)	>0.05
Weight (Kg)	85.00 (32.85)	85.15 (42.70)	85.00 (20.55)	>0.05
Height (cm)	151.8 (12.5)	152.8 (14.3)	148.5 (5.8)	>0.05
BMI	37.0 (16.9)	38.4 (17)	37.0 (12.7)	>0.05
SBP (mm/Hg)	120 (9)	120 (10)	120 (11)	>0.05
DBP (mm/Hg)	80 (10)	80 (10)	80 (10)	>0.05
Triglycerides (mg/dl)	92(64)	97(67)	79(38)	>0.05
Total Chol.(mg/dl)	183.0 (50.3)	183.5 (52.0)	182.0 (48.3)	>0.05
HDL Chol. (mg/dl)	49.0 (20.8)	50.0 (19.0)	42.0 (25.3)	>0.05
LDL Chol. (,mg/dl)	117 (44.5)	118 (46.0)	116 (42.3)	>0.05
HOMA	1.76 (2.09)	2.08 (2.09)	1.76 (2.98)	>0.05
Diabetes mellitus (yes/no)	19/72	13/61	6/11	>0.05
Metabolic Syndrome (yes/no)	36/55	30/44	6/11	>0.05

AIM

To establish whether PWS SGA subjects, defined on the basis of neonatal PWS's percentiles, have a greater risk of developing MetS.

METHOD

We evaluated the presence of the components of the MetS in 91 PWS subject (32 males), aged 27.6 (5.3) years, with a BMI of 37.6 (16.9); 69 (76%, 28 males) had DEL15 and 22 (24%, 4 males) mUPD15. The patients were divided into two groups according their birth weight SDS (BW-SDS): SGA (BW-SDS <-1.5; n=17), appropriate for gestational age (AGA) (BW-SDS -0.682 / +1.5; n=74). In accordance with the literature (Alberti et al., Circulation, 2009; 120: 1640-5), we defined subjects with MetS as having three abnormal findings out of the following five parameters: central obesity, high systolic BP and/or diastolic BP, high triglycerides, low HDL-C and raised fasting plasma glucose. We reported median (IQR) of continuous variables. Statistical analysis was performed by chi square and Mann-Whitney test.



RESULTS

On overall SGA and AGA groups showed similar BMI, weight, length and MetS frequency (40%). The prevalence of MetS in SGA-born obese subjects (7/17; 41%) was similar to that of AGA-born obese (28/74; 38%). Obese girls with DEL15 showed significantly lower frequency of MetS than obese girls with mUPD15 (41% vs 67%; p<0.05).

CONCLUSIONS

Birth weight does not seem to significantly affect the frequency of obesity and MetS in young adults with PWS. Obese females with DEL15, who typically have a shorter birth length than mUPD15, appear to have a lower risk of developing MetS. These data would suggest a direct correlation between length at birth and metabolic risk.

REFERENCES

- Alberti KG, Eckel RH, Grundy SM, et al. Harmonizing the metabolic syndrome: a joint interim statement of the International Diabetes Federation Task Force on Epidemiology and Prevention; National Heart, Lung, and Blood Institute; American Heart Association; World Heart Federation; International Atherosclerosis Society; and International Association for the Study of Obesity. Circulation. 2009 Oct 20;120(16):1640-5.
- Brambilla P, Crinò A, Bedogni G, et al. Metabolic syndrome in children with Prader-Willi syndrome: the effect of obesity. Nutr Metab Cardiovasc Dis. 2011 Apr;21(4):269-76.
- Grugni G, Crinò A, Bedogni G, et al. Metabolic syndrome in adult patients with Prader-Willi syndrome. Nutr Metab Cardiovasc Dis. 2013 Nov;23(11):1134-40.
- Salvatoni A, Moretti A, Grugni G, et al. Anthropometric characteristics of newborns with Prader-Willi syndrome. Am J Med Genet A. 2019 Oct;179(10):2067-2074.

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