Ai sensi dell'art. 3.3 del Regolamento applicativo dell'Accordo Stato-Regioni 05.11.2009, dichiaro che negli ultimi due anni ho avuto i seguenti rapporti in merito a partecipazioni a congressi ed eventi:

- ABBOTT
- MENARINI DIAG.
- ROCHE
- MEDTRONIC
- SANOFI
- LILLY
- NOVO

In fede, Elisabetta Torlone



OBESITA' DIABETE E GRAVIDANZA

Elisabetta Torlone
MISEM A.O. U. di Perugia

Tracking the Global Obesity Epidemic

- Worldwide, the rate of obesity has nearly doubled since 1980: Just over 200 million adult men and just under 300 million adult women have obesity[a]

The increase in the prevalence of overweight and obesity has resulted in more women being obese at the onset of pregnancy, which is associated with a range of adverse outcomes for both mother and child.

a. Finucane MM, et al. *Lancet*. 2011;337:557-567.
b. Ng M, et al. *Lancet*. 2014; 384:766-781.
c. de Onis M, et al. *Am J Clin Nutr*. 2010;92:1257-1264.

Inflammatory and Other Biomarkers: Role in Pathophysiology and Prediction of Gestational Diabetes Mellitus

Pregnancy: an altered inflammatory profile compared to the non-pregnant state, with a fine balance between pro- and anti-inflammatory cytokines needed for normal development. Physiological regulation of the innate immune response and changes in cytokine production prevent rejection of the foetal allograft throughout pregnancy

Int. J. Mol. Sci. **2015**, *16*, 13442-13473

GDM is linked to the down-regulation of adiponectin and anti-inflammatory cytokines (e.g., IL-4 and IL-10) and up-regulation of leptin and pro-inflammatory cytokines implicated in insulin resistance (e.g., IL-6 and TNF-α).

Altered adipokine secretion contributes to glucose homeostasis in pregnancy by both direct and indirect mechanisms

Sally KA, and coll

Catalano P., and coll

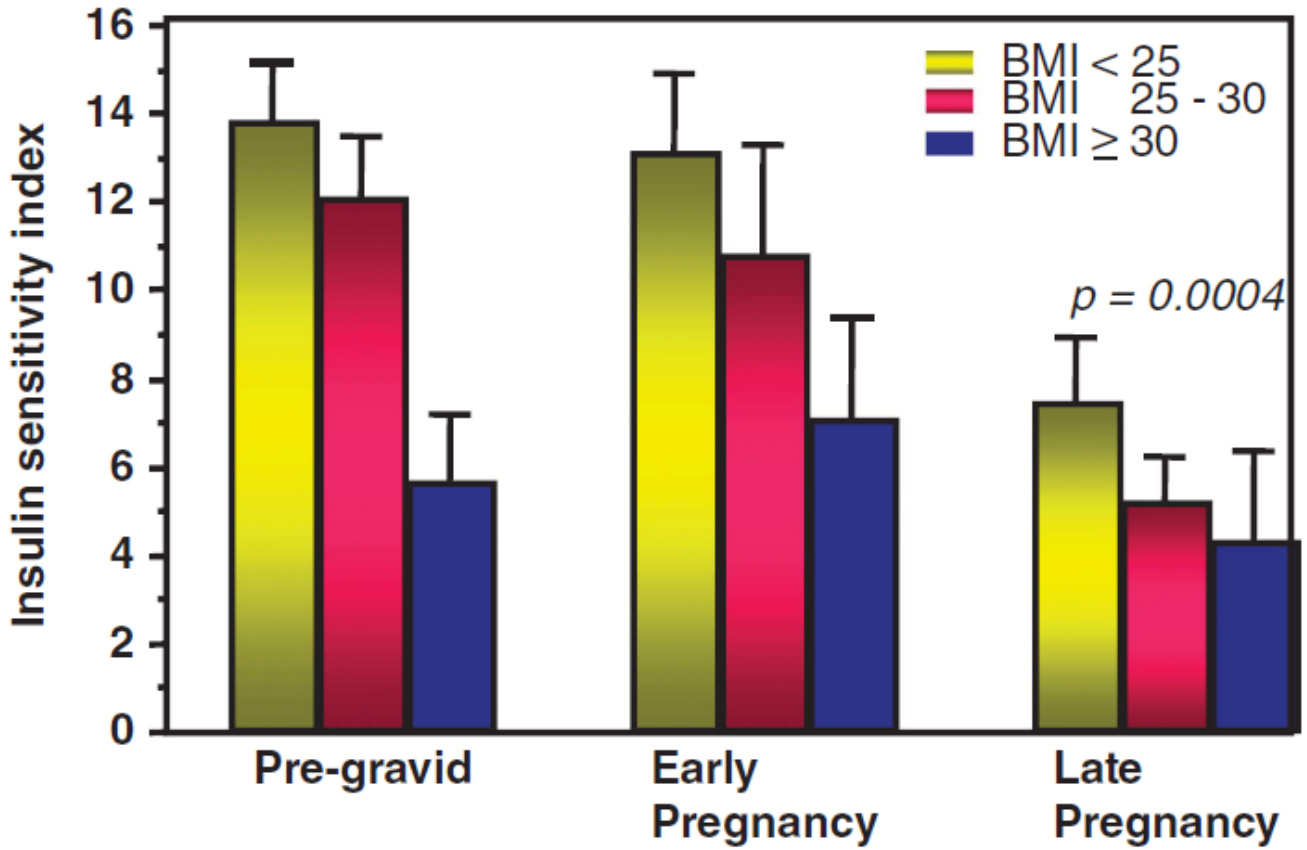


Figure 1. The longitudinal changes in insulin sensitivity, pregravid, early pregnancy (12–14 weeks) and late pregnancy (34–36 weeks) as estimated using the hyperinsulinemic–euglycemic clamp in normal weight, overweight and obese women.[44]

International Journal of Obesity (2015) **39,** 642–649; doi:10.1038/ijo.2015.15

Centre for Maternal and Child Enquiries
Improving the health of mothers, babies and children

CMACE

Royal College of Obstetricians and Gynaecologists
Setting standards to improve women's health

CMACE/RCOG Joint Guideline

Management of Women with Obesity in Pregnancy

APPENDIX 3: Maternal and fetal risks in women with a BMI ≥30 kg/m^2 compared to women with a healthy BMI

Risk	Study	Pop.	Odds ratio [95% Confidence interval]*
Gestational diabetes	NW Thames 1989 – 97 [1]	287213	3.6 [3.3-4.0] [a]
	Aberdeen 1976 – 2005 [2]	24241	2.4 [2.2-2.7]
Hypertensive disorders	NW Thames 1989 – 97 [1]	287213	2.1[1.9-2.5] [a]
	Aberdeen 1976 – 2005 [2]	24241	3.3 [2.7-3.9]
Venous thromboembolism	Denmark 1980 – 2001 [3]	71729	9.7 [3.1-30.8]
Slower labour progress 4 – 10cm	USA 1995 – 2002 [4]	612	7 versus 5.4 hrs p<0.001
Caesarean	Meta-analysis of 33 studies		2.1 [1.9-2.3]
Emergency caesarean	NW Thames 1989 – 97 [1]	287213	1.8 [1.7-1.9]
	Cardiff 1990 – 99 [5]	8350	2.0 [1.2-3.5]
Postpartum haemorrhage	NW Thames 1989 – 97 [1]	287213	1.4 [1.2-1.6] [a]
	Aberdeen 1976 – 2005 [2]	24241	2.3 [2.1-2.6]
Wound infection	NW Thames 1989 – 97 [1]	287213	2.24 [1.91-2.64] [a]
Birth defects	Australia [6]	11252	1.6 [1.0-2.5]
Prematurity	Aberdeen 1976 – 2005 [2]	24241	1.2 [1.1-1.4]
	Australia 1998 – 2002 [6]	11252	1.2 [0.8-1.7]
Macrosomia	NW Thames 1989 – 97 [1]	287213	2.4 [2.2-2.5] [a]
	Sweden 1992 – 2001 [7]	805275	3.1 [3.0-3.3] [b]
Shoulder dystocia	Sweden 1992-2001 [7]	805275	3.14 [1.86-5.31] [b]
	Cardiff 1990 – 99 [5]	8350	2.9 [1.4-5.8]
Admission to NNU	NW Thames 1989 – 97 [1]	287213	1.3 [1.3-1.4] [a]
	Cardiff 1990 – 99 [5]	8350	1.5 [1.1-2.3]
Stillbirth	Meta-analysis of 9 studies [8]		2.1 [1.5-2.7]
Neonatal death	Denmark 1989 – 96 [9]	24505	2.6 [1.2-5.8]

[a] 99% Confidence intervals
[b] OR for morbidly obese
* Unless otherwise stated

OBESITY AND RISK OF GESTATIONAL DIABETES observational studies

	Sebire 2001 (UK)	Weiss 2003 (USA)	Leung 2008 (CINA)	Athukorala 2010 (AUS)	Ovesen 2011 (DENM)	Chung 2012 (CAL)
Overw.	OR 1.68	-	OR 1.75	RR 1.21	OR 3.50	OR 1.74
Obesity	OR 3.60	OR 2.50	OR 2.72	RR 2.10	OR 7.70	OR 2.83
Morbid obesity	-	OR 3.20	OR 4.18	-	OR 11.0	OR 4.72

Risk of major congenital malformations in relation to maternal overweight and obesity severity: cohort study of 1.2 million singletons

Cite this as: *BMJ* 2017;357:j2563

Martina Persson,[1,2] Sven Cnattingius,[1] Eduardo Villamor,[1,3] Jonas Söderling,[1] Björn Pasternak,[1,4] Olof Stephansson,[1,5] Martin Neovius[1]

ABSTRACT

OBJECTIVE

To estimate the risks of major congenital malformations in the offspring of mothers who are underweight (body mass index (BMI) <18.5), overweight (BMI 25 to <30), or in obesity classes I (BMI 30 to <35), II (35 to <40), or III (≥40) compared with offspring of normal weight mothers (BMI 18.5 to <25) in early pregnancy.

DESIGN

Population based cohort study.

SETTING

Nationwide Swedish registries.

PARTICIPANTS

1 243 957 liveborn singleton infants from 2001 to 2014 in Sweden. Data on maternal and pregnancy characteristics were obtained by individual record linkages.

EXPOSURE

Maternal BMI at the first prenatal visit.

MAIN OUTCOME MEASURES

Offspring with any major congenital malformation, and subgroups of organ specific malformations diagnosed during the first year of life. Risk ratios were estimated using generalised linear models adjusted for maternal factors, sex of offspring, and birth year.

Risk of major congenital malformations in relation to maternal overweight and obesity severity: cohort study of 1.2 million singletons

Martina Persson,[1,2] Sven Cnattingius,[1] Eduardo Villamor,[1,3] Jonas Söderling,[1] Björn Pasternak,[1,4] Olof Stephansson,[1,5] Martin Neovius[1]

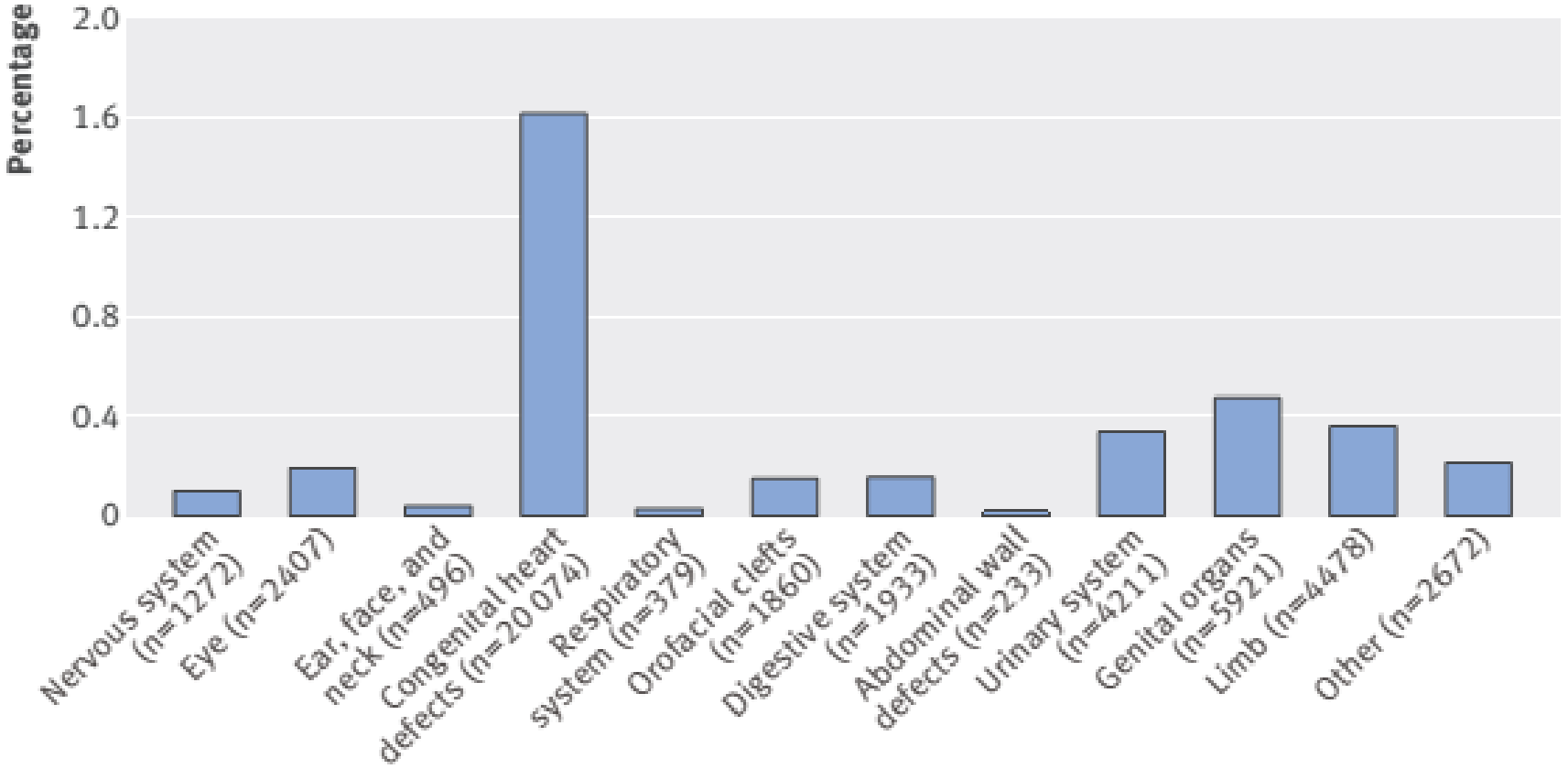


Fig 1 | Prevalence of major congenital malformations in swedish live singleton births between 2001 and 2014 (n =1 243 957)

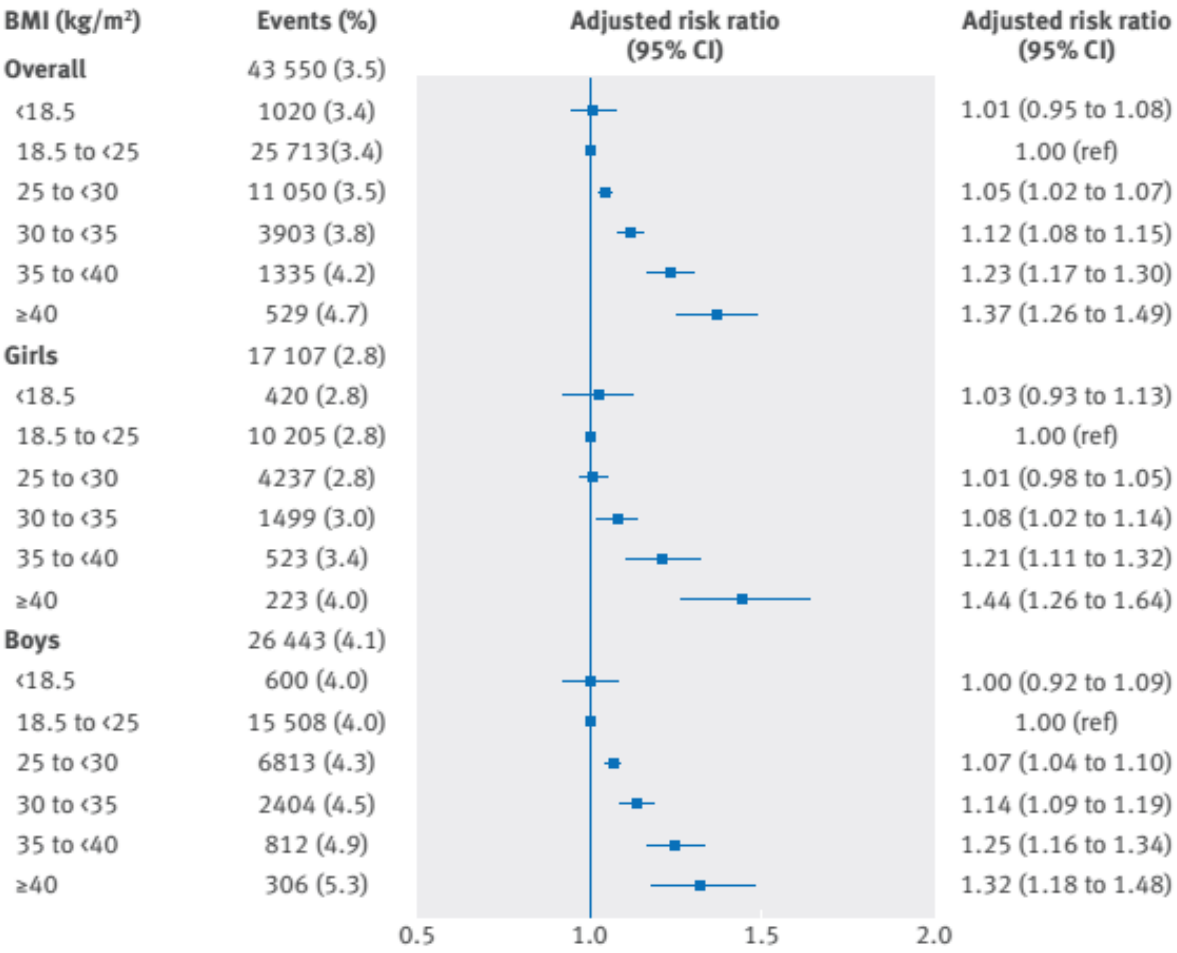


Fig 2 | Major congenital malformations in liveborn singletons by maternal body mass index (BMI) in underweight (BMI <18.5; n=29 864), normal weight (BMI 18.5 to <25; n=756 432), and overweight (BMI 25 to <30; n=311 339) women, and in women in obesity classes I (BMI 30 to <35; n=103 085), II (BMI 35 to <40; n=31 883), and III (BMI ≥40; n=11 354). Adjustment was made for maternal age (13-24, 25-29, 30-34, ≥35 years), height (130-154, 155-159, 160-164, 165-169, 170-174, 175-200 cm), parity (primiparous, multiparous), early pregnancy smoking status (non-smoker, 1-9, ≥10 cigarettes daily), educational level (<10, 10-12, >12 years), maternal country of birth (Nordic (Sweden, Denmark, Finland, Iceland, and Norway), non-Nordic), family situation (living with partner, not living with partner), and sex of offspring

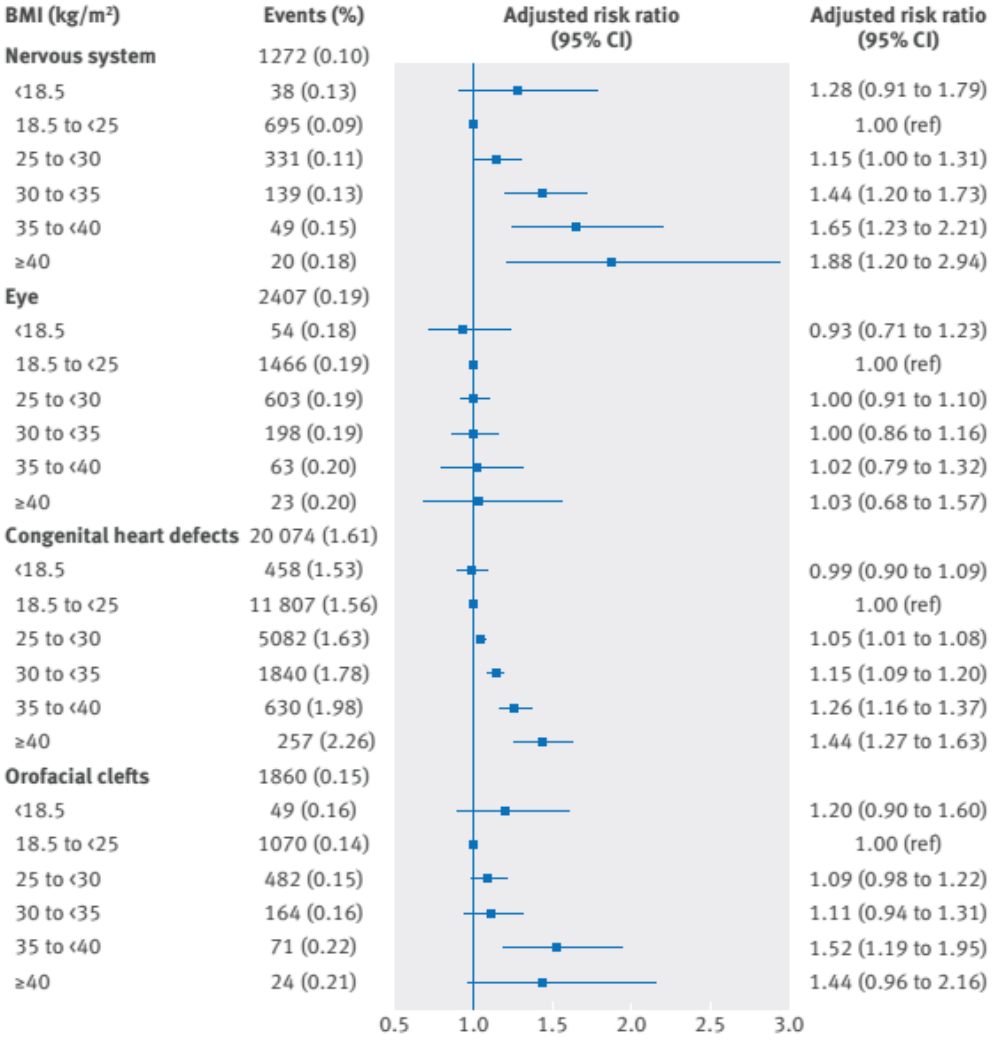


Fig 3 | Major congenital malformations in nervous system, eye, heart, and oral clefts in liveborn singletons by maternal body mass index (BMI) in underweight (BMI <18.5; n=29 864), normal weight (BMI 18.5 to <25; n=756 432), and overweight (BMI 25 to <30; n=311 339) women, and in women in obesity classes I (BMI 30 to <35; n=103 085), II (BMI 35 to <40; n=31 883), and III (BMI ≥40; n=11 354). Adjustment was made for maternal age (13-24, 25-29, 30-34, ≥35 years), height (130-154, 155-159, 160-164, 165-169, 170-174, 175-200 cm), parity (primiparous, multiparous), early pregnancy smoking status (non-smoker, 1-9, ≥10 cigarettes daily), educational level (<10, 10-12, >12 years), maternal country of birth (Nordic (Sweden, Denmark, Finland, Iceland, and Norway), non-Nordic), family situation (living with partner, not living with partner), and sex of offspring

Maternal obesity during pregnancy and cardiovascular development and disease in the offspring

Table 2 Adverse offspring outcomes of maternal obesity during pregnancy

Fetal outcomes	Childhood outcomes	Adult outcomes
Stillbirth	Obesity	Obesity
Neonatal death	Adverse body composition	Increased blood pressure
Congenital anomalies	Increased blood pressure	Adverse lipid profile
Large size for gestational age at birth	Adverse lipid profile	Impaired insulin/glucose homoeostasis
Neonatal hypoglycemia	Increased inflammatory markers	Premature mortality
Referral to neonatal intensive care unit	Impaired insulin/glucose homoeostasis	

Cardiovascular and metabolic consequences during fetal life, childhood and adulthood of maternal prepregnancy obesity or excessive gestational weight gain

Gaillard R. Eur J Epidemiol (2015) 30:1141–1152

Maternal Obesity, Gestational Weight Gain, and Offspring Adiposity: The Exploring Perinatal Outcomes among Children Study

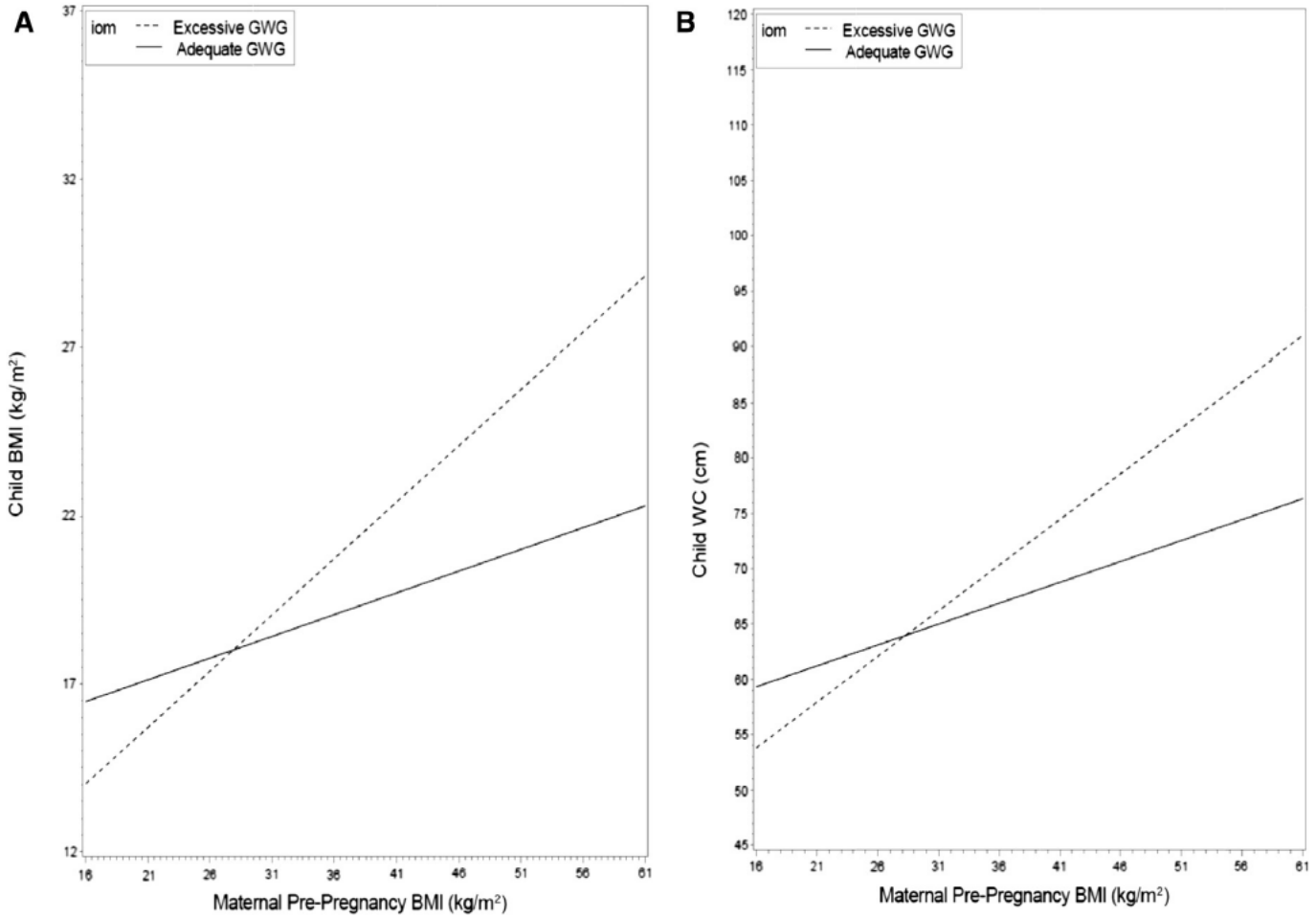
Objective To determine whether adequate vs excessive gestational weight gain (GWG) attenuated the association between maternal obesity and offspring outcomes.

Study design Data from 313 mother-child pairs participating in the Exploring Perinatal Outcomes among Children study were used to test this hypothesis. Maternal prepregnancy body mass index (BMI) and weight measures throughout pregnancy were abstracted from electronic medical records. GWG was categorized according to the 2009 Institute of Medicine criteria as adequate or excessive. Offspring outcomes were obtained at a research visit (average age 10.4 years) and included BMI, waist circumference (WC), subcutaneous adipose tissue (SAT) and visceral adipose tissue, high-density lipoprotein cholesterol, and triglyceride levels.

Jill L. Kaar, PhD[1], Tessa Crume, PhD[2], John T. Brinton, PhD[3], Kimberly J. Bischoff, MSHA[4], Robert McDuffie, MD[5], and Dana Dabelea, MD, PhD[2]

THE JOURNAL OF PEDIATRICS

Maternal Obesity, Gestational Weight Gain, and Offspring Adiposity: The Exploring Perinatal Outcomes among Children Study



Jill L. Kaar, PhD[1], Tessa Crume, PhD[2], John T. Brinton, PhD[3], Kimberly J. Bischoff, MSHA[4], Robert McDuffie, MD[5], and Dana Dabelea, MD, PhD[2]

THE JOURNAL OF PEDIATRICS

Maternal Obesity, Gestational Weight Gain, and Offspring Adiposity: The Exploring Perinatal Outcomes among Children Study

Results. More overweight/obese mothers exceeded the Institute of Medicine GWG recommendations (68%) compared with normal-weight women (50%) (P < .01). Maternal prepregnancy BMI was associated with worse childhood outcomes, particularly among offspring of mothers with excessive GWG (increased BMI [20.34 vs 17.80 kg/m2], WC [69.23 vs 62.83 cm], SAT [149.30 vs 90.47 cm2], visceral adipose tissue [24.11 vs 17.55 cm2], and homeostatic model assessment [52.52 vs 36.69], all P < .001).

The effect of maternal prepregnancy BMI on several childhood outcomes was attenuated for offspring of mothers with adequate vs excessive GWG (P < .05 for the interaction between maternal BMI and GWG status on childhood BMI, WC, SAT, and high-density lipoprotein cholesterol).



The Hyperglycemia and Adverse Pregnancy Outcome Study

Associations of GDM and obesity with pregnancy outcomes

Patrick M. Catalano, MD[1]
H. David McIntyre, MD[2]
J. Kennedy Cruickshank, MD[3]
David R. McCance, MD[4]
Alan R. Dyer, PHD[5]
Boyd E. Metzger, MD[6]
Lynn P. Lowe, PHD[5]
Elisabeth R. Trimble, MD[7]
Donald R. Coustan, MD[8]
David R. Hadden, MD[4]
Bengt Persson, MD, PHD[9]
Moshe Hod, MD[10]
Jeremy J.N. Oats, MD[11]
for the HAPO Study Cooperative Research Group

Diabetes Care 35:780–786, 2012

- **Entrambi GDM e obesità materna sono indipendentemente associati con outcomes avversi.**
- **La combinazione dei due, comunque, ha un maggiore impatto che uno solo di essi.**



P. Catalano et al.
HAPO Study

Diabetes Care 35:780–786, 2012

GDM, OBESITY , AND ADVERSE PREGNANCY OUTCOMES

Outcome	N	No.	%	Model I OR	Model I 95% CI	Model II OR	Model II 95% CI
Birth weight >90th percentile[1]							
No GDM, no obesity	17,244	1,339	7.8	1.00		1.00	
GDM, no obesity	2,791	401	14.4	1.99	(1.77–2.25)	2.19	(1.93–2.47)
No GDM, obesity	2,247	278	12.4	1.68	(1.46–1.92)	1.73	(1.50–2.00)
GDM, obesity	935	203	21.7	3.29	(2.79–3.89)	3.62	(3.04–4.32)
Cord C-peptide >90th percentile[2]							
No GDM, no obesity	14,886	916	6.2	1.00		1.00	
GDM, no obesity	2,419	386	16.0	2.90	(2.54–3.30)	2.49	(2.17–2.85)
No GDM, obesity	1,829	201	11.0	1.80	(1.52–2.12)	1.77	(1.49–2.11)
GDM, obesity	751	168	22.4	4.14	(3.43–5.00)	3.61	(2.94–4.42)
Primary cesarean[3]							
No GDM, no obesity	15,673	2,522	16.1	1.00		1.00	
GDM, no obesity	2,442	564	23.1	1.48	(1.33–1.65)	1.25	(1.12–1.40)
No GDM, obesity	1,868	430	23.0	1.80	(1.60–2.03)	1.51	(1.33–1.71)
GDM, obesity	749	215	28.7	2.32	(1.96–2.75)	1.71	(1.44–2.04)
Preeclampsia[4]							
No GDM, no obesity	16,238	570	3.5	1.00		1.00	
GDM, no obesity	2,518	149	5.9	1.69	(1.40–2.05)	1.74	(1.43–2.11)
No GDM, obesity	1,878	250	13.3	3.47	(2.95–4.08)	3.91	(3.31–4.62)
GDM, obesity	730	147	20.1	5.25	(4.26–6.46)	5.98	(4.81–7.42)
Newborn percent body fat >90th percentile[5]							
No GDM, no obesity	14,367	1,143	8.0	1.00		1.00	
GDM, no obesity	2,338	331	14.2	1.91	(1.67–2.18)	1.98	(1.73–2.27)
No GDM, obesity	1,854	233	12.6	1.66	(1.43–1.93)	1.65	(1.41–1.93)
GDM, obesity	768	185	24.1	3.67	(3.08–4.38)	3.69	(3.06–4.44)
Shoulder dystocia/birth injury[4]							
No GDM, no obesity	17,247	212	1.2	1.00		1.00	
GDM, no obesity	2,792	41	1.5	1.10	(0.78–2.25)	1.14	(0.81–1.62)
No GDM, obesity	2,252	32	1.4	0.99	(0.68–1.92)	1.03	(0.70–1.53)
GDM, obesity	936	26	2.8	1.71	(1.12–3.89)	1.84	(1.19–2.86)

Fetal metabolic programming

This concept was first suggested by Barker and Hales in the early 1990s. They hypothesized that fetal and perinatal events, such as maternal undernutrition, were central to determine one's risk to develop chronic metabolic diseases.

Such conditions, including obesity, diabetes, and cardiovascular diseases, have become a very important population health concern.

It is now clear that the environment can exert a strong influence on the development:
"Developmental plasticity"

Nutritional status during pregnancy has important implications for subsequent maternal and offspring health.
Inadequate intake during this time may have a negative impact on health outcomes in later adult life.

Phenotype of Infants of Mothers with Gestational Diabetes

DiabetesCare

Table 2—*Neonatal body composition and anthropometrics in AGA infants of women with GDM and NGT*

	GDM	*NGT*	*P*
n	132	175	—
Weight (g)	3,202±357	3,249±372	0.27
Fat-free mass (g)	2,832±286	2,919±287	0.008
Fat mass (g)	371±163	329±150	0.02
Body fat (%)	11.4±4.6	9.9±4.0	0.002
Tricep (mm)	4.5±0.9	4.1±0.8	0.0002
Subscapular (mm)	5.1±1.1	4.5±1.0	0.0001
Flank (mm)	4.0±1.2	3.7±0.8	0.007
Thigh (mm)	5.7±1.2	5.2±1.3	0.002
Abdomen (mm)	3.3±0.9	3.0±0.8	0.002

P. CATALANO, A.THOMAS 2007

Phenotype of Infants of Mothers with Gestational Diabetes

DiabetesCare

Table 3—*Neonatal body composition in LGA infants of women with GDM and NGT*

	GDM	NGT	*P*
n	50	52	—
Weight (g)	4,060±380	4,120±351	0.13
Fat-free mass (g)	3,400± 312	3,564±310	0.0009
Fat mass (g)	662±163	563±206	0.02
Body fat (%)	16.2± 3.3	13.5±4.5	0.002

P. CATALANO, A.THOMAS 2007

ORIGINAL ARTICLE

Maternal obesity programs mitochondrial and lipid metabolism gene expression in infant umbilical vein endothelial cells

SMR Costa[1,2,3,4], E Isganaitis[2,3,4], TJ Matthews[2], K Hughes[2], G Daher[2], JM Dreyfuss[2], GAP da Silva[1] and M-E Patti[2,3]

BACKGROUND/OBJECTIVES: Maternal obesity increases risk for childhood obesity, but molecular mechanisms are not well understood. We hypothesized that primary umbilical vein endothelial cells (HUVEC) from infants of overweight and obese mothers would harbor transcriptional patterns reflecting offspring obesity risk.

SUBJECTS/METHODS: In this observational cohort study, we recruited 13 lean (pre-pregnancy body mass index (BMI) $< 25.0\ kg\ m^{-2}$) and 24 overweight-obese ('ov-ob', BMI $\geqslant 25.0\ kg\ m^{-2}$) women. We isolated primary HUVEC, and analyzed both gene expression (Primeview, Affymetrix) and cord blood levels of hormones and adipokines.

RESULTS: A total of 142 transcripts were differentially expressed in HUVEC from infants of overweight-obese mothers (false discovery rate, FDR < 0.05). Pathway analysis revealed that genes involved in mitochondrial and lipid metabolism were negatively correlated with maternal BMI (FDR < 0.05). To test whether these transcriptomic patterns were associated with distinct nutrient exposures in the setting of maternal obesity, we analyzed the cord blood lipidome and noted significant increases in the levels of total free fatty acids (lean: $95.5 \pm 37.1\ \mu g\ ml^{-1}$, ov-ob: $124.1 \pm 46.0\ \mu g\ ml^{-1}$, $P = 0.049$), palmitate (lean: $34.5 \pm 12.7\ \mu g\ ml^{-1}$, ov-ob: $46.3 \pm 18.4\ \mu g\ ml^{-1}$, $P = 0.03$) and stearate (lean: $20.8 \pm 8.2\ \mu g\ ml^{-1}$, ov-ob: $29.7 \pm 17.2\ \mu g\ ml^{-1}$, $P = 0.04$), in infants of overweight-obese mothers.

CONCLUSIONS: Prenatal exposure to maternal obesity alters HUVEC expression of genes involved in mitochondrial and lipid metabolism, potentially reflecting developmentally programmed differences in oxidative and lipid metabolism.

International Journal of Obesity advance online publication, 4 October 2016; doi:10.1038/ijo.2016.142

Table 2. Newborn characteristics and cord blood adipokines according to maternal weight category

Newborn data	*BMI < 25.0 kg m^{-2}*	*BMI ⩾ 25.0 kg m^{-2}*	P-*value*
Gestational age (weeks)	39.1 ± 1.3 (13)	39.1 ± 1.3 (24)	0.85
Birth weight (g)	3280 ± 662 (13)	3229 ± 508 (24)	0.81
Infant sex (M/F)	10M/3F	13M/11F	0.28
Weight for gestational age and sex (percentile)	41.5 ± 27.1 (13)	45.2 ± 26.5 (24)	0.69
Head circumference (cm)	33.9 ± 1.3 (13)	34.3 ± 2.1 (24)	0.56
Abdominal girth (cm)	31.6 ± 3.0 (13)	31.6 ± 2.0 (24)	0.96
Length (cm)	48.9 ± 2.8 (13)	48.8 ± 2.7 (24)	0.93
Ponderal index (kg m^{-3})	27.7 ± 2.4 (13)	27.6 ± 2.6 (24)	0.88
Cord blood hormones and adipokines			
Insulin (μU ml^{-1})	4.6 ± 3.9 (12)	9.0 ± 9.1 (24)	**0.048**
HGF (ng ml^{-1})	2.5 ± 1.0 (12)	3.1 ± 1.0 (24)	0.08
Leptin (ng ml^{-1})	23.9 ± 19.4 (13)	49.0 ± 43.6 (24)	**0.02**
Adiponectin (μg ml^{-1})	407 ± 377 (13)	337 ± 296 (24)	0.57
Resistin (pg ml^{-1})	174 ± 97 (12)	196 ± 110 (24)	0.55
IL-1β (pg ml^{-1})	0.12 ± 0.06 (12)	0.17 ± 0.29 (23)	0.50
IL-6 (pg ml^{-1})	11 ± 10 (12)	41.9 ± 77.7 (23)	0.07
IL-8 (pg ml^{-1})	61 ± 137 (12)	85 ± 210 (24)	0.68
MCP-1 (pg ml^{-1})	386 ± 245 (13)	314 ± 205 (23)	0.38
TNF-α (pg ml^{-1})	9.1 ± 2.5 (12)	10.8 ± 3.7 (24)	0.11
Total PAI-1 (pg ml^{-1})	186 ± 62 (13)	152 ± 32 (23)	0.08

Abbreviations: BMI, body mass index; F, female; HGF, hepatocyte growth factor; IL, interleukin; M, male; MCP-1, monocyte chemoattractant protein 1; PAI-1, plasminogen activator inhibitor 1; TNF-α, tumor necrosis factor alpha. Mean ± s.d.; sample size is indicated in parentheses. *P*-value refers to Student's *t*-test, except for infant sex, which was analyzed by Fisher's exact test. Bold indicate significant *P*-values.

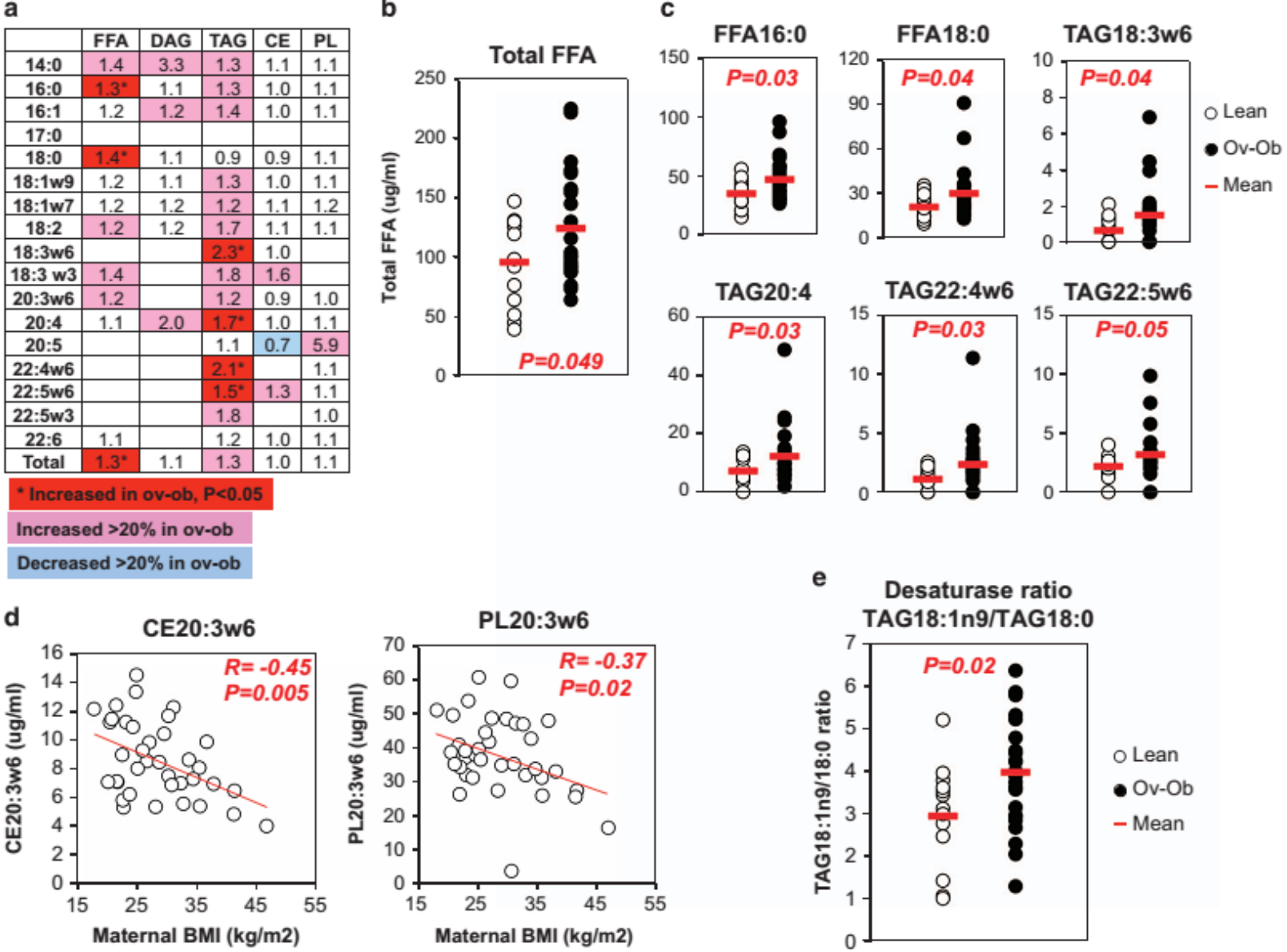


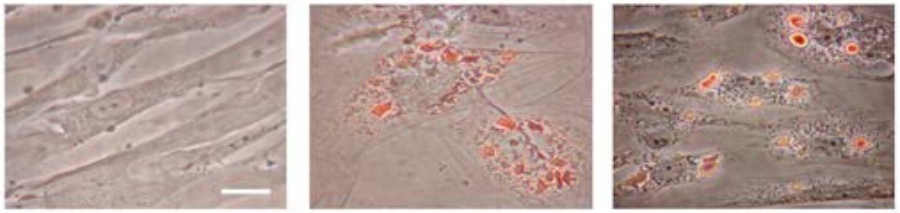
Figure 3. Maternal obesity alters infant cord blood lipidome. (**a**) Overview of lipid composition of cord blood. Each column represents a class of lipids, and each row represents a fatty acid comprised within the lipid class. Each lipid subset was quantified by liquid chromatography/mass spectrometry (LC/MS); then normalized to the level in infants of lean mothers. Data are expressed as fold-change in ov-ob/lean. CE, cholesterol esters; DAG, diacylglycerides; FFA, free fatty acids; PL, phospholipids; TAG, triacylglycerides. (**b**) Cord blood total fatty acids (μg ml^{-1}); $N = 37$. (**c**) Cord blood FFA16:0, FFA18:0, omega-6 conjugated triglycerides and TAG20:4 are increased in infants of overweight/obese women. All lipid levels are expressed as μg ml^{-1}; $N = 37$. (**d**) 20:3w6-conjugated cholesterol esters and phospholipids are inversely correlated with maternal BMI; $N = 37$. (**e**) The ratio of 18:1n9 to 18:0 triglycerides, a surrogate for stearoyl-coA desaturase 1 (*SCD1*) activity, is increased in cord blood from infants of ov-ob women; $N = 37$.

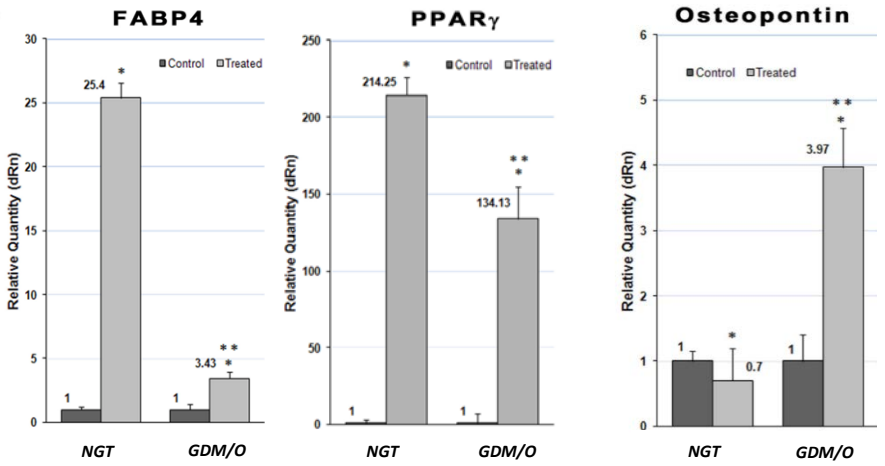
We compared hUCMS stemness and immunoregulatory features of **GDM and obese** (n=20), well monitored during pregnancy, *vs.* **normal weight and euglycemic** (n=10) mothers

- **hUCMS plasticity** (differentiation into other cell phenotypes)
- **hUCMS immunomodulatory properties *in vitro***

hUCMS plasticity differentiation pathways

Adipogenic differentiation





Osteogenic differentiation



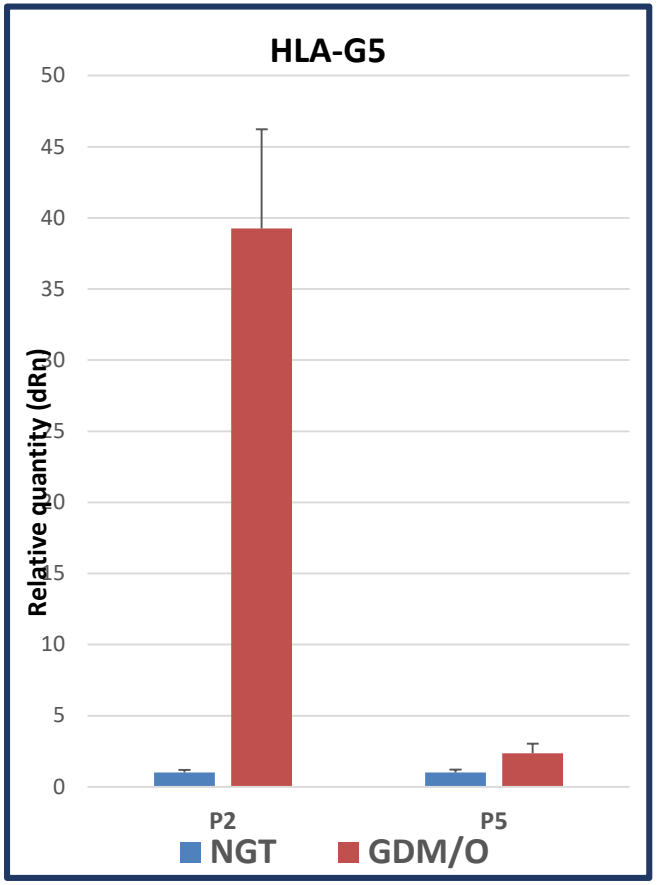
Expression of immunomodulatory markers

qPCR for immunomodulatory markers

		IDO 1	iNOS	HLAG1	HLAG5
hUCMS NGT	II	0,000	0,000	0,000	1,000
	III	1,000	0,000	0,000	3,569
	IV	3,160	0,000	0,000	2,643
	V	1,940	0,000	0,000	23,300
		IDO 1	**iNOS**	**HLAG1**	**HLAG5**
hUCMS GDM/O	II	1,000	1,000	1,000	1,000
	III	0,080	0,025	0,275	0,086
	IV	0,072	0,071	0,143	0,026
	V	0,482	0,043	0,000	1,490

Relative quantity (dRn)

Expression of each marker, relative to its own control, equal 1



QUANDO DOBBIAMO INTERVENIRE?

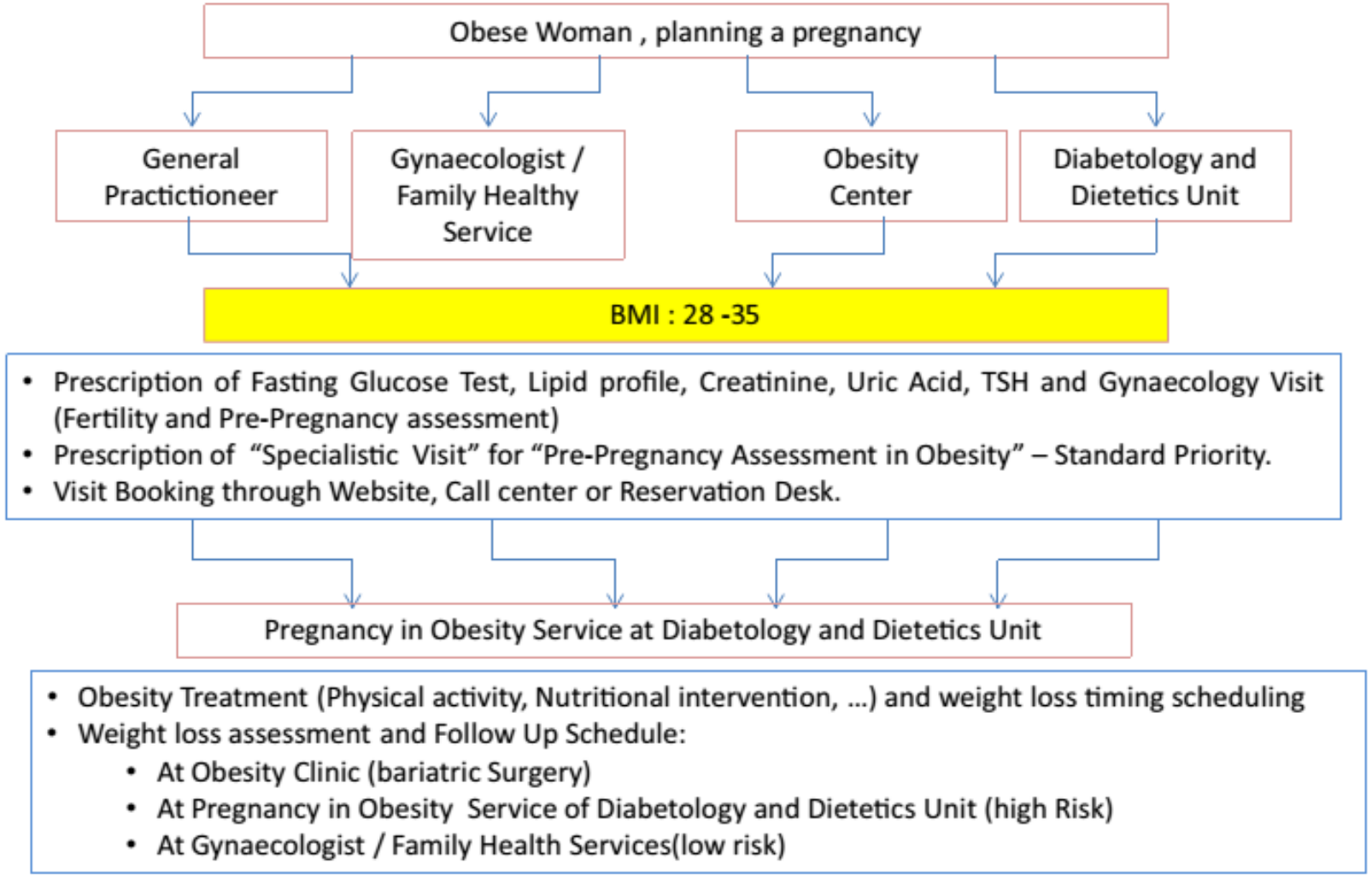


Fig. 2 Clinical pregestational pathway for pregnancy in obesity

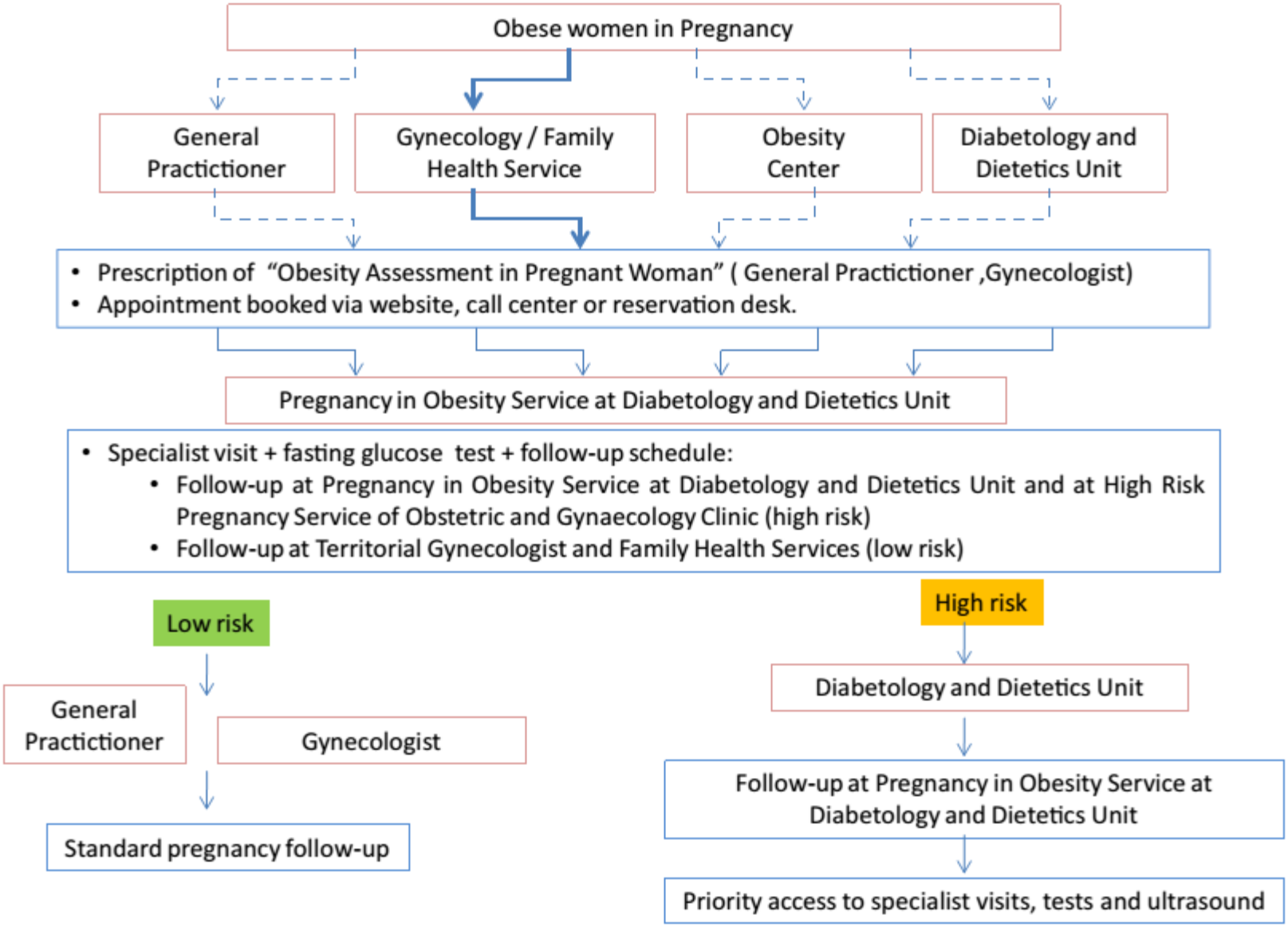


Fig. 1 Clinical gestational pathway for pregnancy in obesity

TAKE HOME MESSAGES

-Obesità e GDM in gravidanza devono essere considerata entrambe condizioni di elevato rischio sia per la madre che per il bambino
-E' importante definire dei percorsi strutturati per la riduzione del peso nelle gestanti obese prima della gravidanza e, se non possibile, per un adeguato incremento ponderale nel corso della gravidanza sia delle obese che delle donne con GDM
-Gli obiettivi attuali devono essere ambiziosi perché solo un lavoro intensivo può portare a migliorare gli outcomes fetali e a investire adeguatamente sulla salute futura.



GRAZIE