Bambino Gesù
OSPEDALE PEDIATRICO

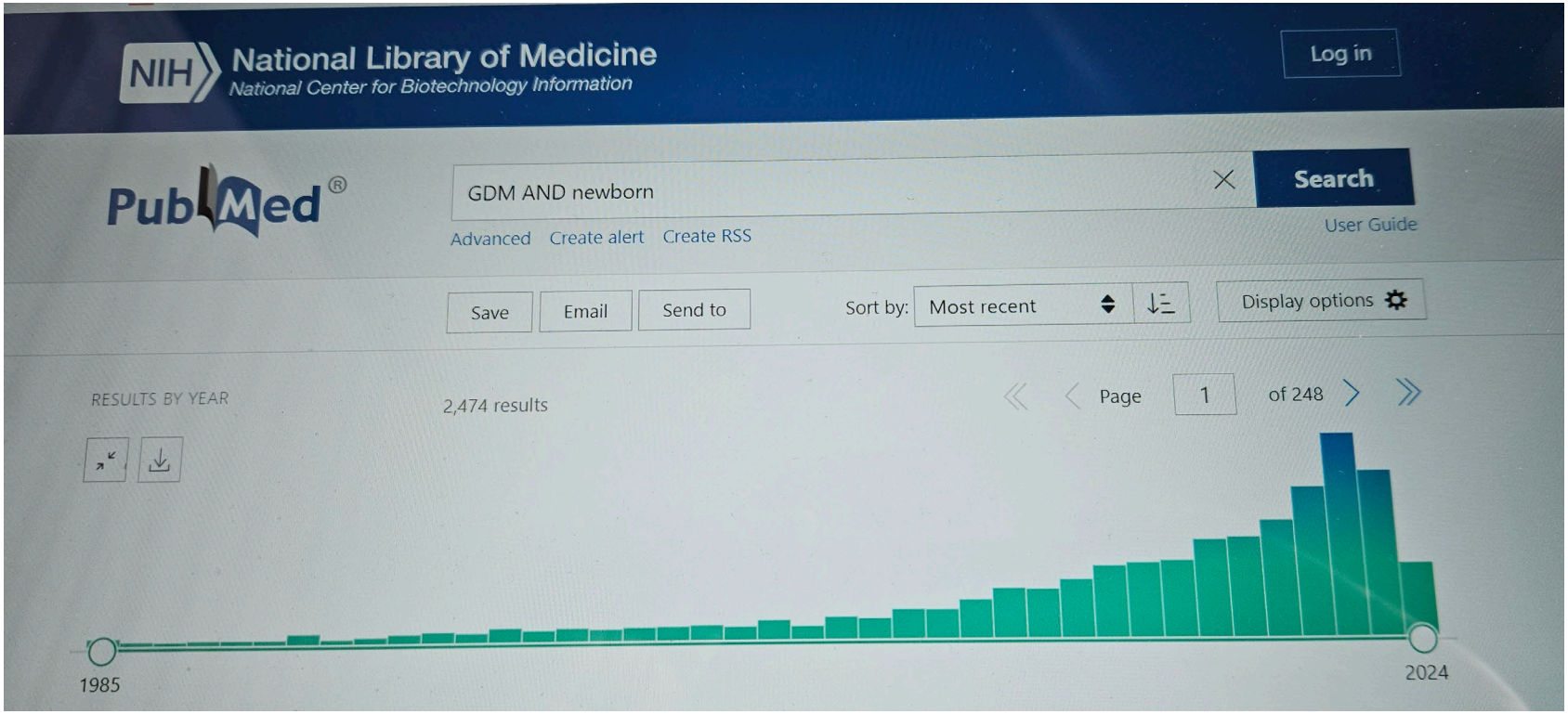
Andrea Dotta
Terapia Intensiva Neonatale
Ospedale Pediatrico Bambino Gesù
Roma

Il neonato di madre diabetica

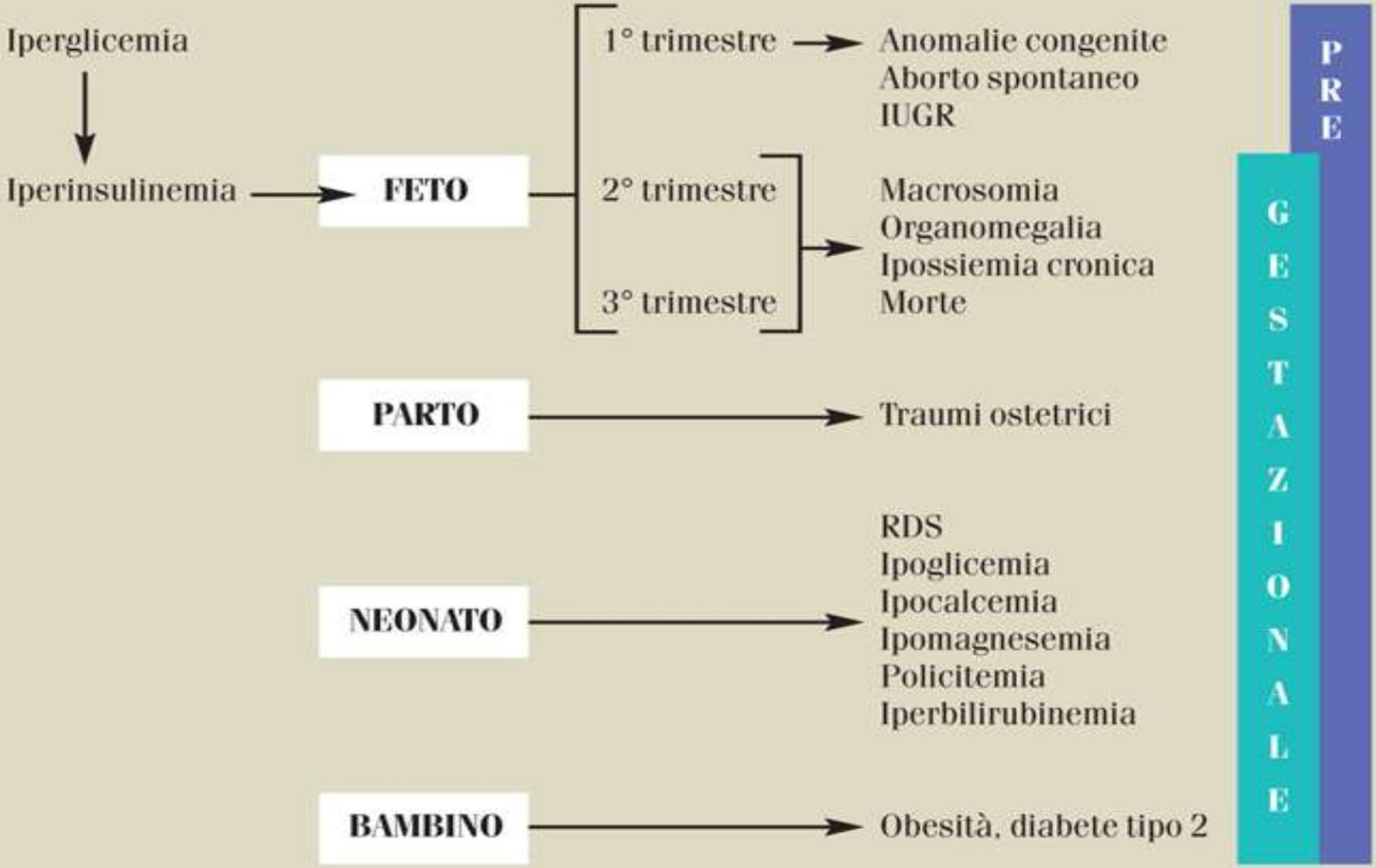
Il Dr Andrea Dotta dichiara di aver ricevuto negli ultimi due anni compensi o finanziamenti dalle seguenti Aziende Farmaceutiche e/o Diagnostiche:

- Astra Zeneca
- Sanofi

Dichiara altresì il proprio impegno ad astenersi, nell'ambito dell'evento, dal nominare, in qualsivoglia modo o forma, aziende farmaceutiche e/o denominazione commerciale e di non fare pubblicità di qualsiasi tipo relativamente a specifici prodotti di interesse sanitario (farmaci, strumenti, dispositivi medico-chirurgici, ecc.).

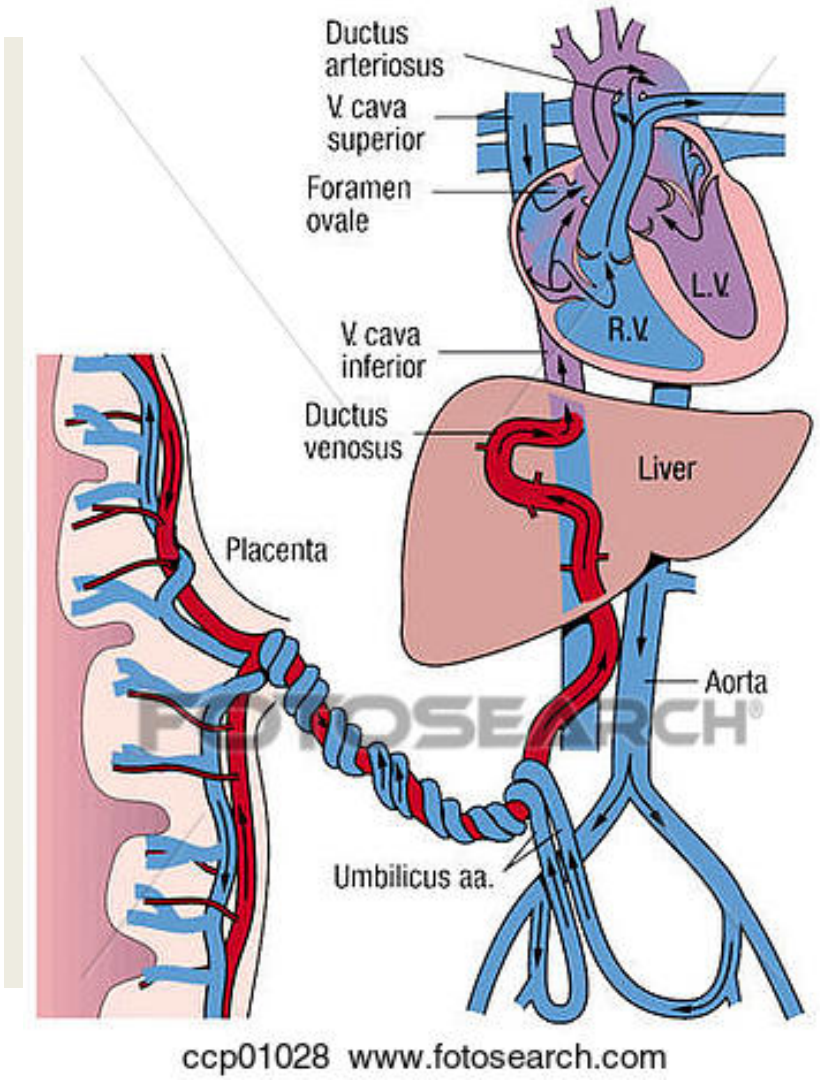

NIH
National Library of Medicine
National Center for Biotechnology Information
Log in
PubMed®
GDM AND newborn
Search
Advanced
Create alert
Create RSS
User Guide
Save
Email
Send to
Sort by:
Most recent
Display options
RESULTS BY YEAR
2,474 results
Page
1
of 248
1985
2024

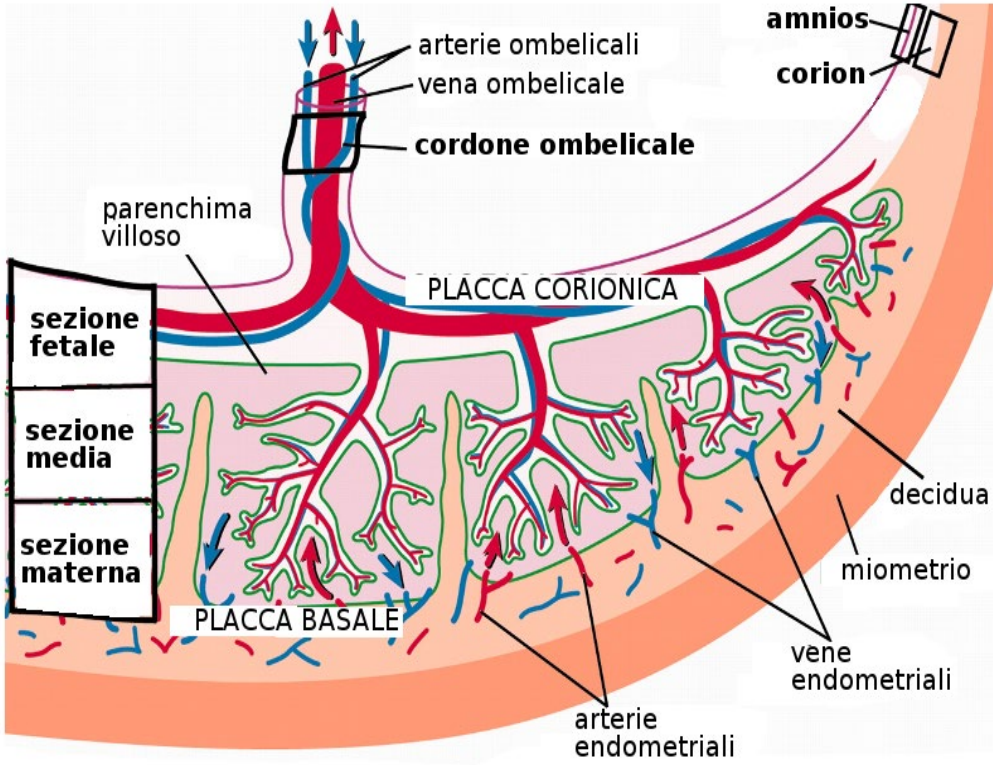

io
speriamo
che me
la cavo



Fonte: Aloraini IA. Pictorial Essay: Infants of Diabetic Mothers. Indian J Radiol Imaging. 2010; 20(3): 174-81

La placenta



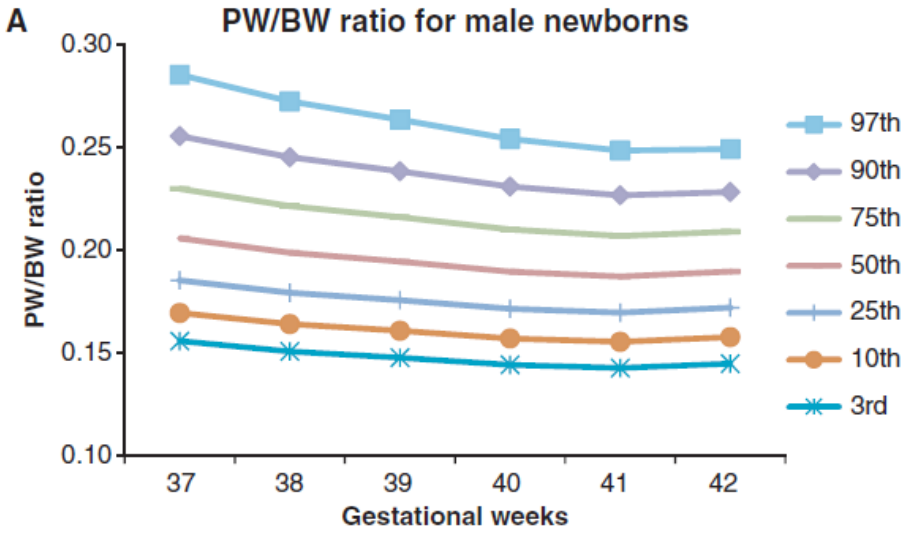


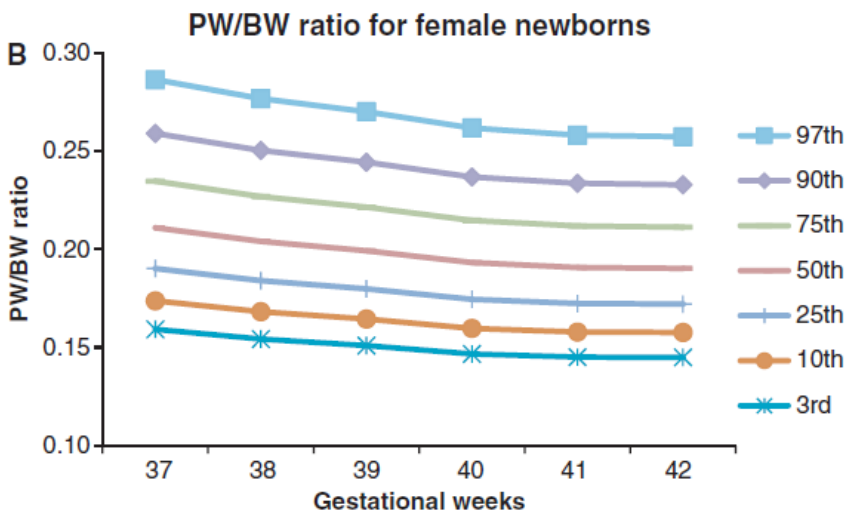
F. Gloria-Bottini et al. 2015

andrea.dotta@opbg.net

Placenta/birthweight ratio and perinatal outcome: a retrospective cohort analysis

F Shehata,[a] I Levin,[b] A Shrim,[a] B Ata,[a] B Weisz,[c] R Gamzu,[b] B Almog[a]

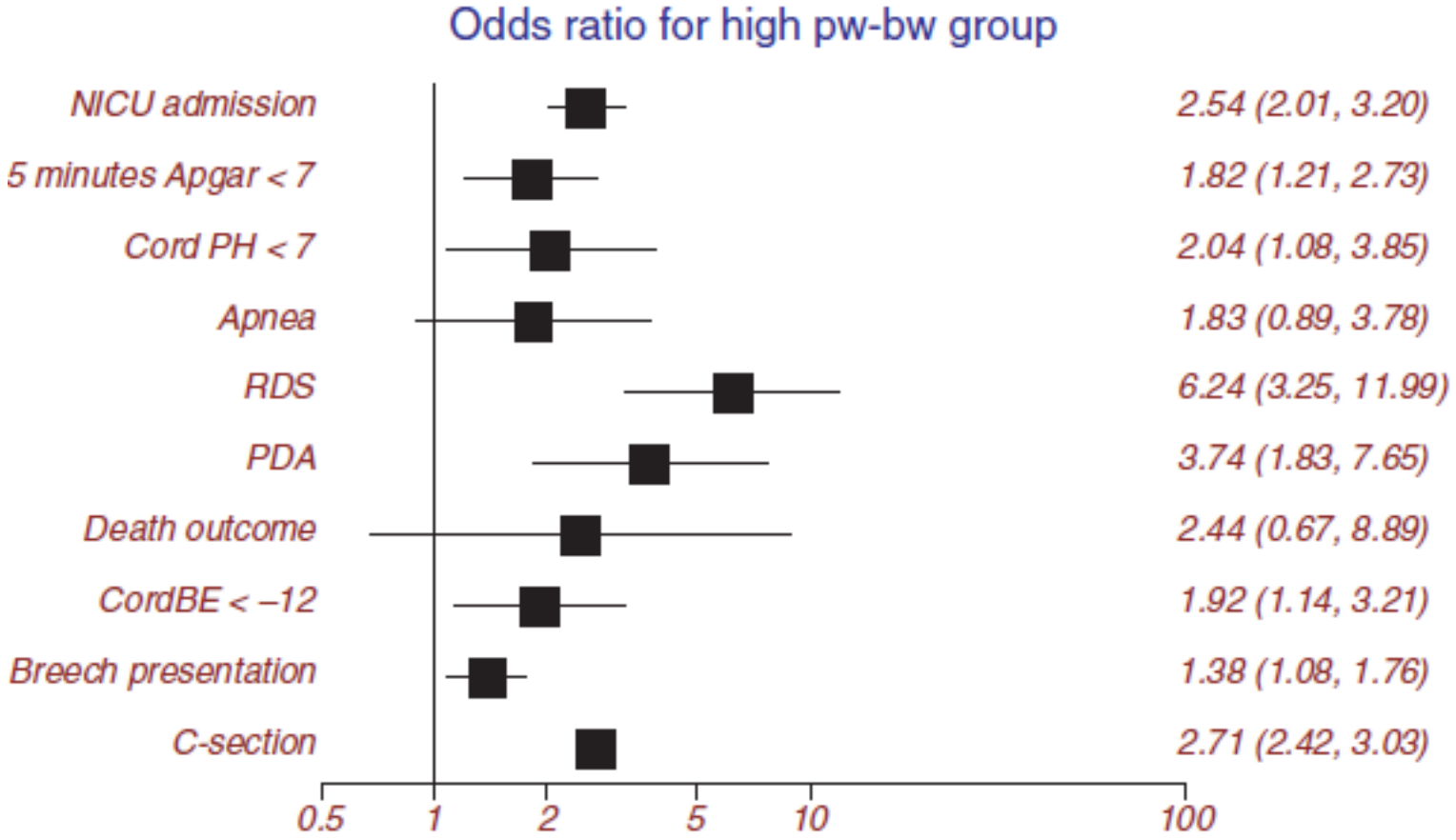




DOI: 10.1111/j.1471-0528.2011.02892.x
www.bjog.org

andrea.dotta@opbg.net

Placenta/birthweight ratio and perinatal outcome: a retrospective cohort analysis



DOI: 10.1111/j.1471-0528.2011.02892.x
www.bjog.org

andrea.dotta@opbg.net

The Placental Role in Gestational Diabetes Mellitus: A Molecular Perspective

DOI: https://doi.org/10.17925/EE.2024.20.1.5

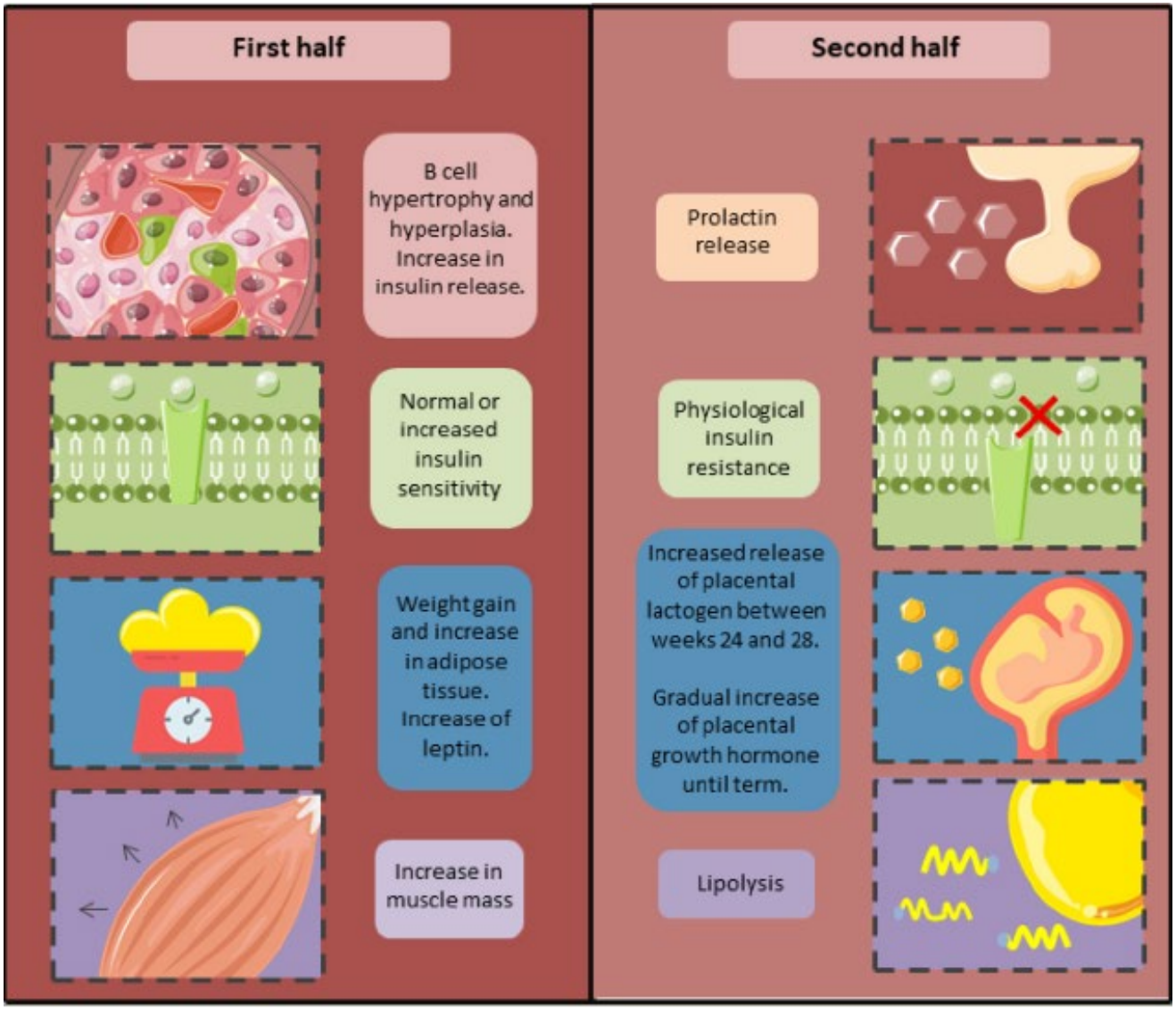


Figure 1: Physiological changes in macromolecule metabolism during pregnancy[16]

Journal Publication Date: 30 April 2024

The Placental Role in Gestational Diabetes Mellitus: A Molecular Perspective

DOI: https://doi.org/10.17925/EE.2024.20.1.5

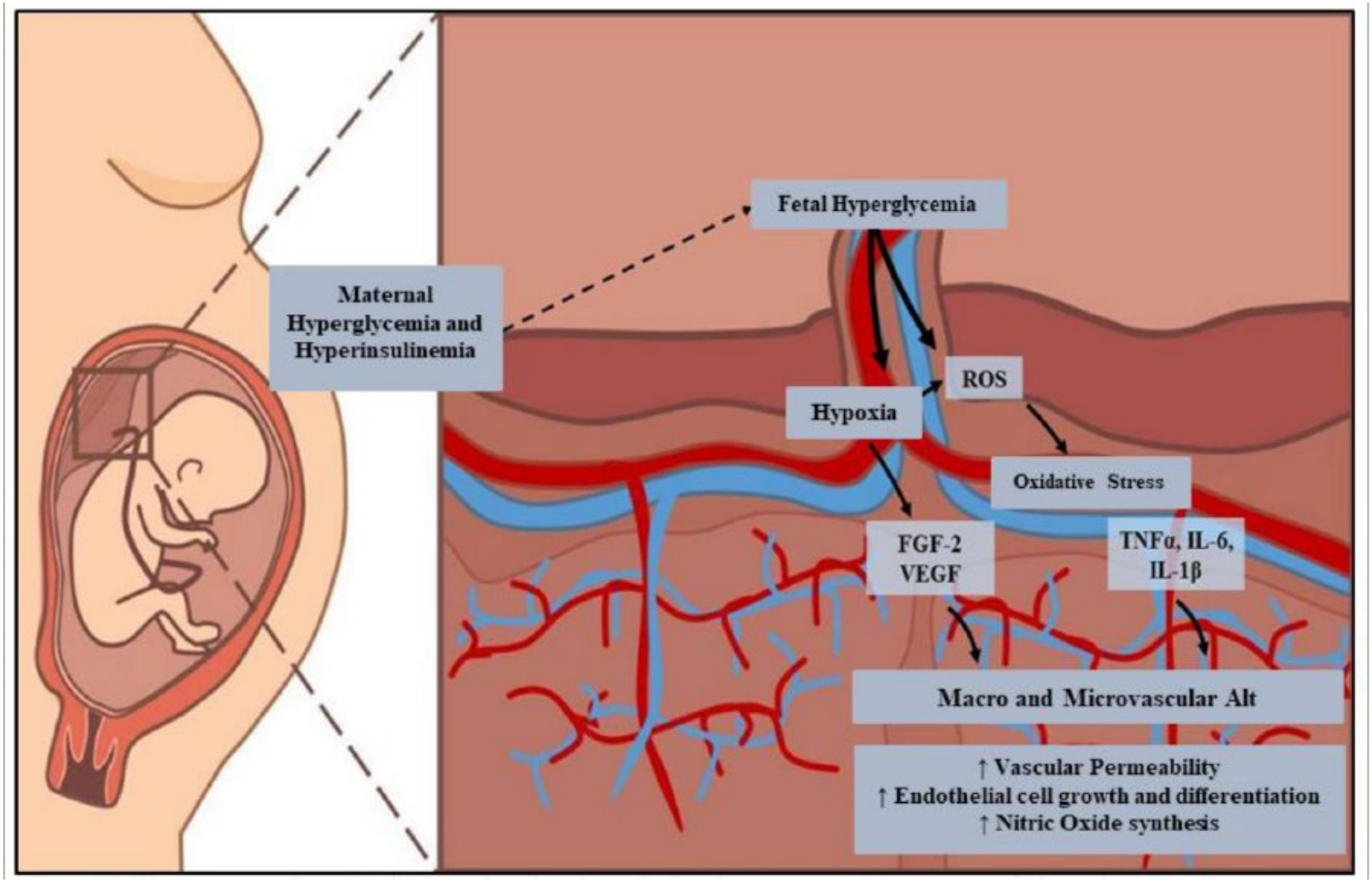
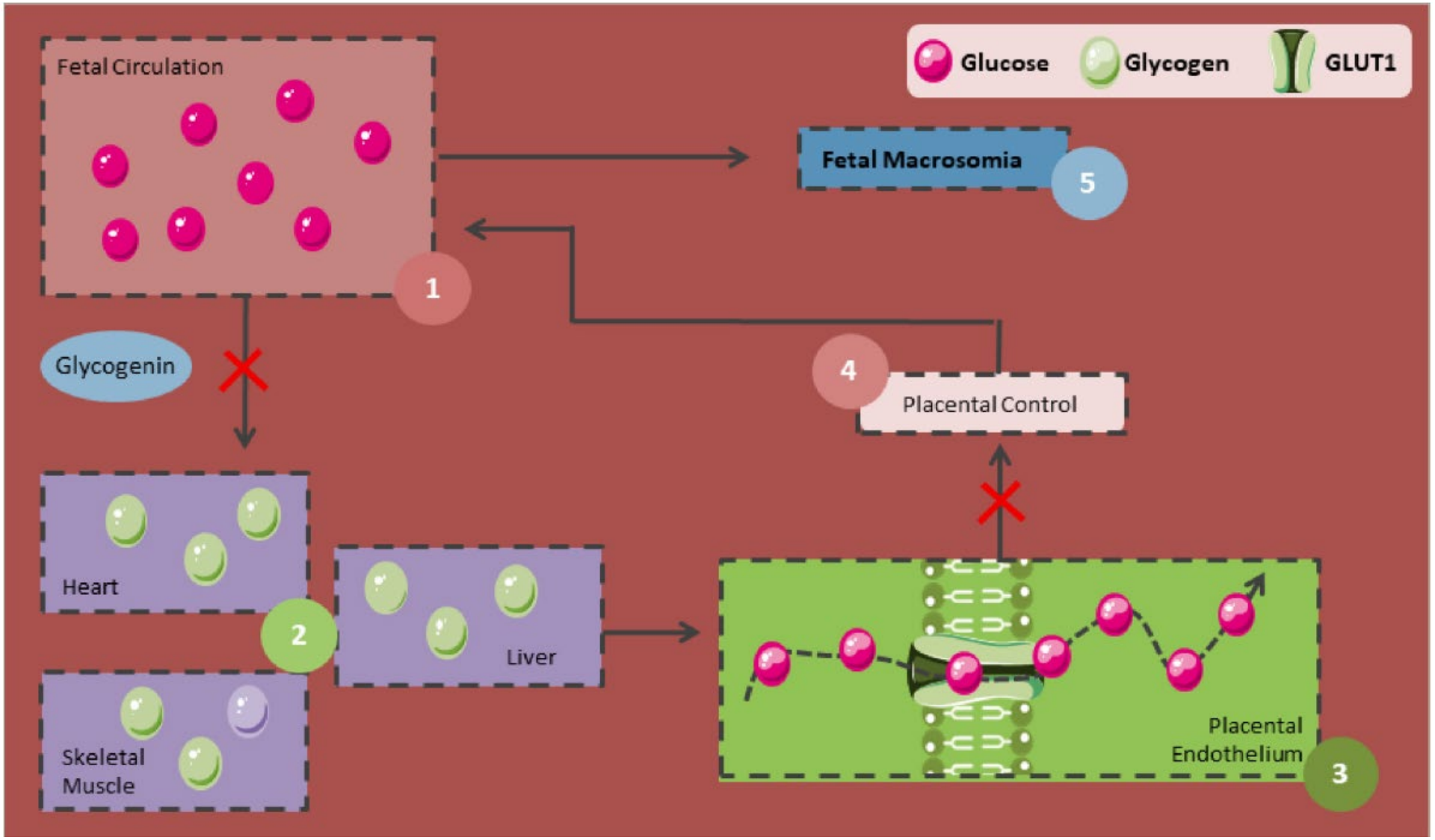


Figure 3: The macrovascular and microvascular alterations of the placenta occur due to maternal hyperglycaemia and compensatory hyperinsulinaemia, which lead to foetal hyperglycaemia

The Placental Role in Gestational Diabetes Mellitus: A Molecular Perspective

DOI: https://doi.org/10.17925/EE.2024.20.1.5



Physiopathology of foetal macrosomia

The Placental Role in Gestational Diabetes Mellitus: A Molecular Perspective

DOI: https://doi.org/10.17925/EE.2024.20.1.5

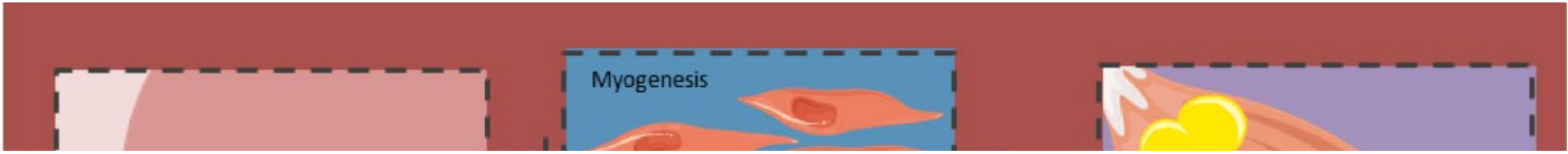
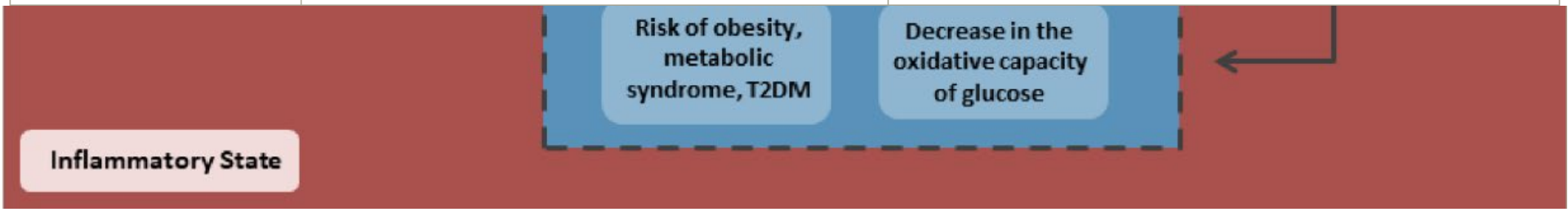


Table 2: Hormonal disturbances and alterations in placental transport and growth occurring in gestational diabetes mellitus and the impact on the foetus

Feature	Women without GDM	Proposed pathophysiological effects on the foetus
Hormones	Elevated insulin (insulin resistance) Impaired glucose tolerance Elevated adipokines	Foetal overgrowth (increased risk of macrosomia) Risk of foetal hypoglycaemia due to increased insulin secretion
Placental growth	Abnormalities in placental growth and vascular development Reduced placental perfusion	Foetal hypoxia Foetal growth restriction contributing to IUGR
Placental transport	Altered nutrient and oxygen transfer	Foetal developmental delay IUGR Increased risk of metabolic abnormalities



Decrease in the oxidative capacity of skeletal muscle

Non-hypertensive gestational diabetes mellitus: Placental histomorphology and its association with perinatal outcomes

Histomorphological alterations in GDM placentas compared with the Non-GDM placentas.

Histological Features		GDM n = 54 (%)	Non-GDM n = 33 (%)	P Value
Chronic inflammation	No inflammation	45 (83.3)	33 (100.0)	**0.031***
	Chronic villitis	8 (14.8)	0 (0.0)	
	Chronic deciduitis	1 (1.9)	0 (0.0)	
Maternal vascular malperfusion	No MVM	32 (59.3)	31 (93.9)	**0.003***
	Decidual vasculopathy	14 (25.9)	1 (3.0)	
	AVM/DVH	6 (11.1)	1 (3.0)	
	Increased fibrin deposition	1 (1.9)	0 (0.0)	
	Villous infarction	1 (1.9)	0 (0.0)	
Syncytial knots/villous (%)		42.8 ± 20.8	33.2 ± 12.9	**0.020***
Fetal vascular malperfusion	No FVM	51 (94.4)	33 (100.0)	0.703
	Avascular villi	2 (3.7)	0 (0.0)	
	Subintimal fibrin deposition	1 (1.9)	0 (0.0)	
Villous dysmaturity	Yes	9 (16.7)	0 (0.0)	**0.012***
	No	45 (83.3)	33 (100.0)	
Chorangiosis	Yes	10 (18.5)	1 (3.0)	**0.046***
	No	44 (81.5)	32 (97.0)	
Others	Villous oedema	12 (22.2)	5 (15.2)	0.886
	Meconium effects	4 (7.4)	2 (6.0)	
	Intervillous haematoma	2 (3.7)	1 (3.0)	
	None of the above	36 (66.7)	25 (75.8)	

https://doi.org/10.1016/j.placenta.2024.01.012 Placenta 147 (2024) 21–27

Non-hypertensive gestational diabetes mellitus: Placental histomorphology and its association with perinatal outcomes

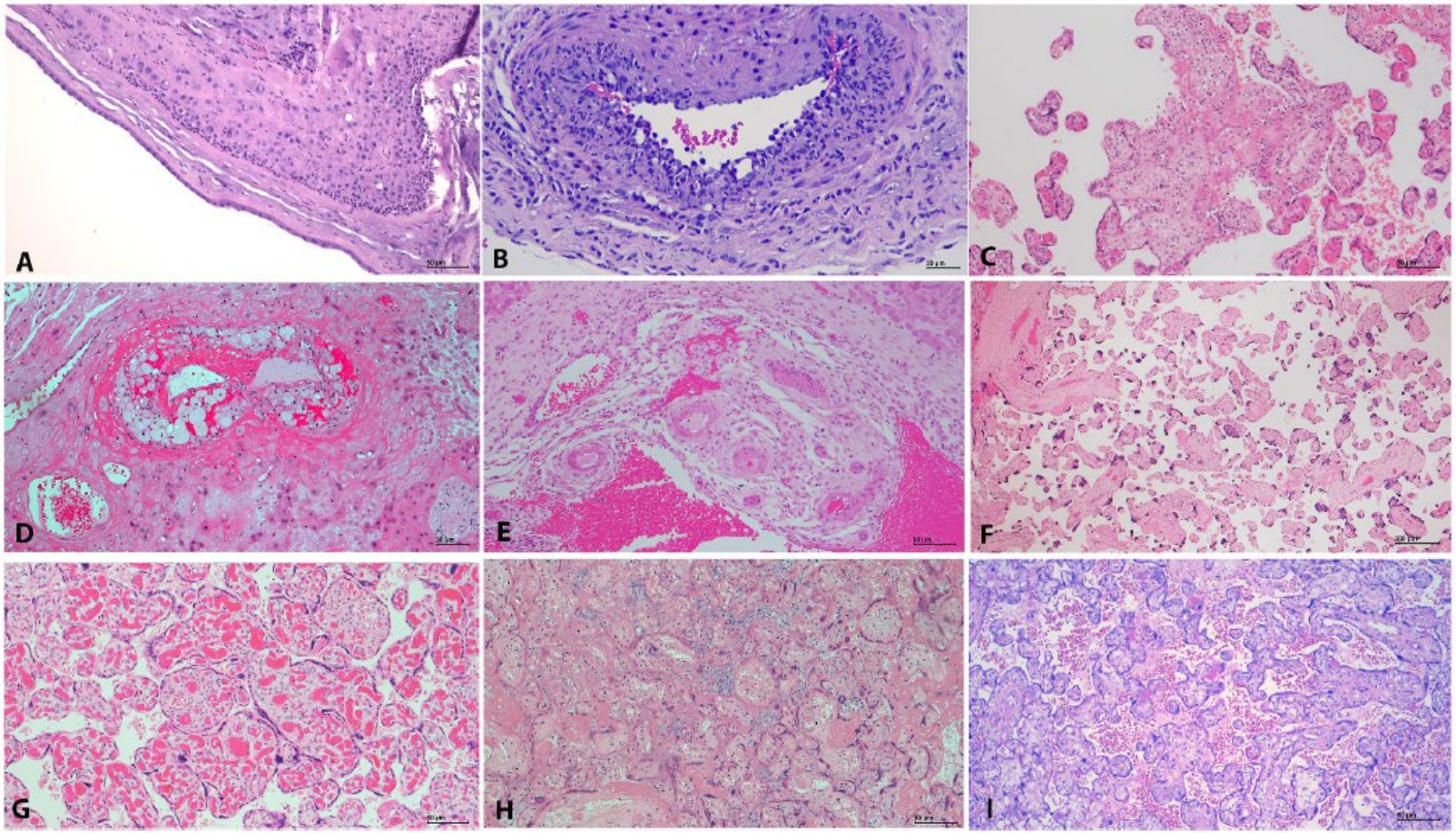


Fig. 2. Histological characteristics of GDM-affected placentas showing (**A**) acute chorioamnionitis with moderate neutrophilic infiltrates (H&E x200), (**B**) chorionic vasculitis (H&E x400), (**C**) chronic low grade villitis (H&E x200), (**D**) acute atherosis of maternal decidual vessel (H&E x400), (**E**) mural hypertrophy of maternal decidual vessels (H&E x200), (**F**) distal villous hypoplasia (H&E x100), (**G**) chorangiosis (H&E x200), (**H**) villous infarction (H&E x200), and (**I**) delayed villous maturation in a term placenta (H&E x200).

https://doi.org/10.1016/j.placenta.2024.01.012

Placenta 147 (2024) 21–27

Gestational diabetes mellitus induces placental vasculopathies

Table 1 Maternal and neonatal characteristics

Characteristic	Control, mean ± SD ($N=40$)	GDM, mean ± SD ($N=44$)	*p*-value
Maternal			
Age (year)	28.2 ± 5.6	31.2 ± 5.5	0.04
Weight (kg)	74.9 ± 13.6	82.7 ± 14.9	< 0.01
Body mass index (kg/m^2)	30.2 ± 4.96	33.2 ± 5.8	0.02
OGTT results (mmol/L)			
Fasting	4.4 ± 0.45	5.03 ± 1.02	< 0.01
1 h	7.04 ± 1.52	10.05 ± 1.66	< 0.01
2 h	5.87 ± 1.46	8.79 ± 1.75	< 0.01
Neonatal			
Gestational age (week)	39.0 ± 1.15	38.5 ± 1.29	0.07
Birth weight (g)	3065 ± 339	3274 ± 421	0.02
Sex, M/F	17/23	20/24	—
Placental weight (g)	421.4 ± 64.3	460.8 ± 79.2	0.02
Umbilical cord length (cm)	52.7 ± 10.14	53.5 ± 12.66	0.69

GDM gestational diabetes mellitus, *OGTT* oral glucose tolerance test, *SD* standard deviation, *M/F* male/female

Environmental Science and Pollution Research (2022) 29:19860–19868
https://doi.org/10.1007/s11356-021-17267-y

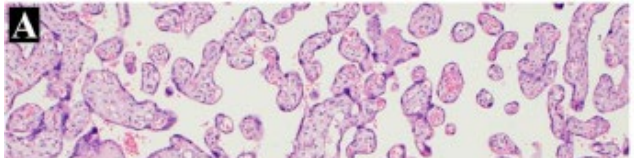
Table 2 Vasculopathies in the maternal side of the placenta

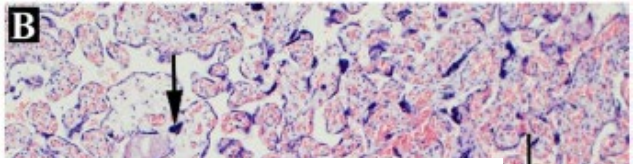
Placental vasculopathy	Control ($N=40$) *N* (%)	GDM ($N=44$) *N* (%)	*p-value*
Decidual vasculopathy	2 (5)	19 (43.2)	< 0.01
Placental infarction	3 (7.5)	3 (6.8)	0.90
Intervillous fibrin	7 (17.5)	10 (22.7)	0.55
Syncytial knots	11 (27.5)	34 (77.3)	< 0.01
Villous agglutination	8 (20)	25 (56.8)	< 0.01
Calcification	16 (40)	31 (70.5)	< 0.01
Retroplacental hemorrhage	0 (0)	15 (34.1)	< 0.01
Primitive cytotrophoblast	17 (42.5)	20 (45.5)	0.79

Table 4 Vasculopathies in the fetal side of the placenta

Placental vasculopathy	Control ($N=40$) *N* (%)	GDM ($N=44$) *N* (%)	*p-value*
Chorangiosis	11 (27.5)	22 (50)	0.04
Uniformly avascular villi	3 (7.5)	3 (6.8)	0.90
Villous fibrinoid necrosis	7 (17.5)	31 (70.5)	< 0.01
Fibromuscular sclerosis	10 (25)	22 (50)	0.02
Villous edema	6 (15)	17 (38.6)	0.02
Obliterative	2 (5)	2 (4.5)	0.92
Subchorionic intervillous thrombohematoma	3 (7.5)	3 (6.8)	0.90
Villous thrombosis	6 (15%)	4 (9.1)	0.40

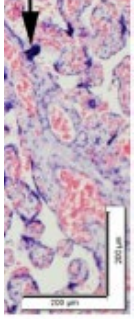
GDM gestational diabetes mellitus

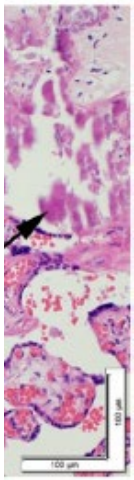




Conclusion

In the GDM group, decidual vasculopathy, syncytial knots, villous agglutination, calcification, retroplacental hemorrhage, chorangiosis, villous edema, villous fibroid necrosis, and fibromuscular sclerosis were significantly increased compared to the control group, indicating that these histopathological findings indicate GDM in the mother. Such findings in the placenta of women who have not been diagnosed with GDM indicate that these women should be screened for GDM in future pregnancies, screened for type 2 diabetes mellitus after pregnancy, and followed up to ensure the well-being of both the mother and baby.





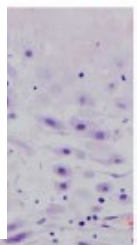
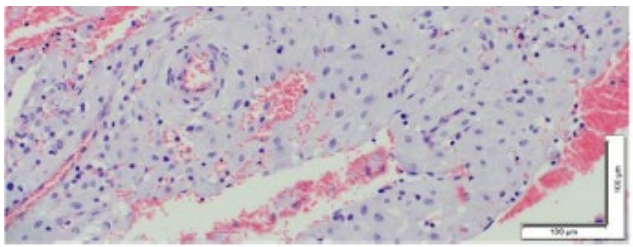
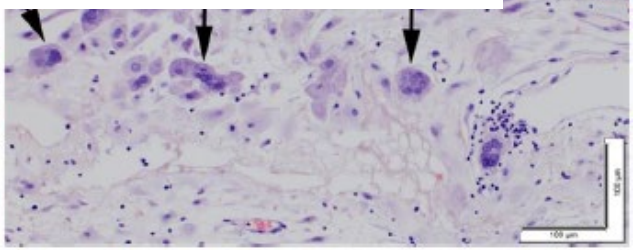
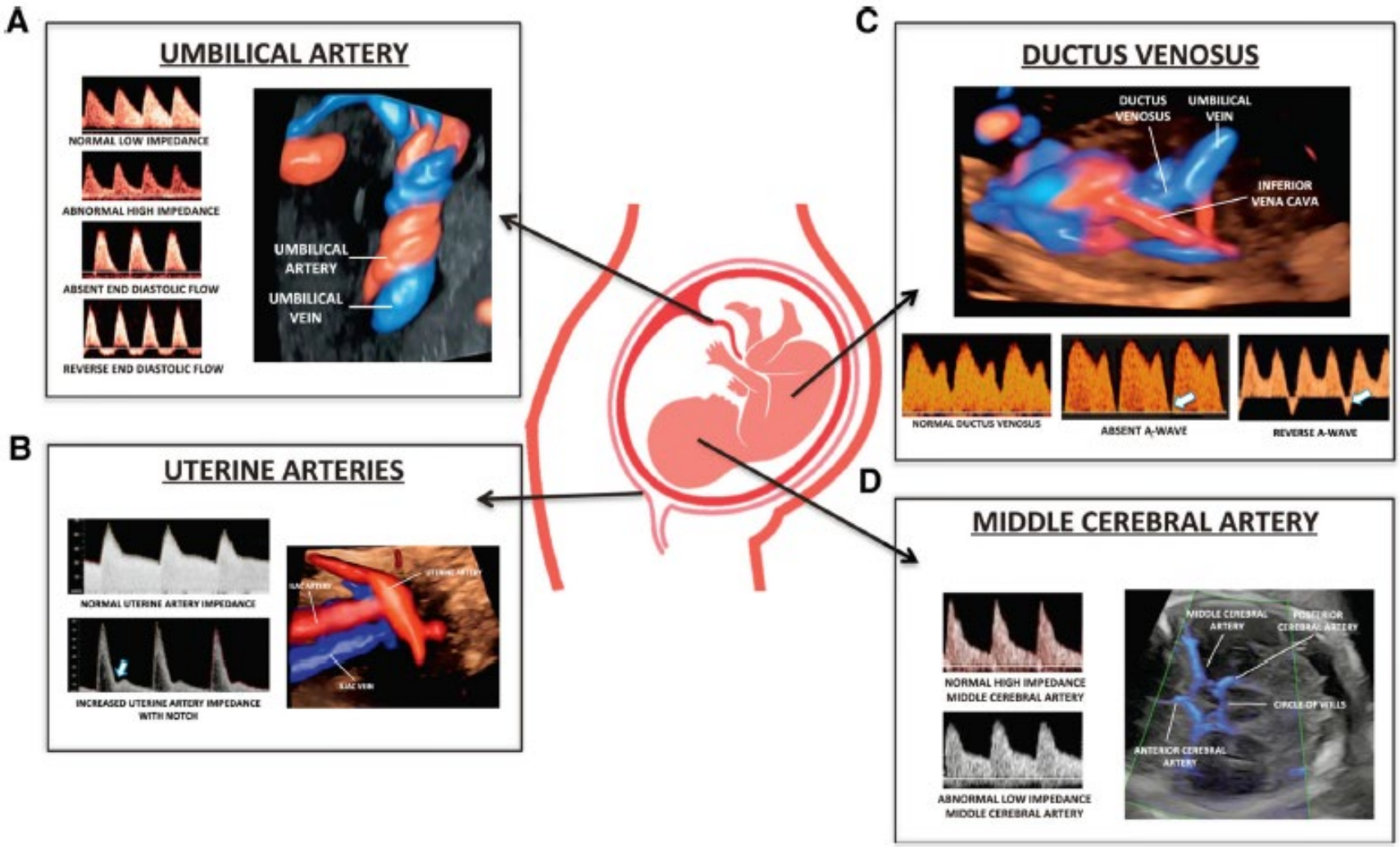


Fig. 1 Mater
changes in th
(**maternal** sid
placenta, ma
B Syncytial k
magnificatio
Intervillous f
villous agglu
cation 200×.
(arrows), mag
Decidual vas
magnificatio
cytotrophobla
magnificatio



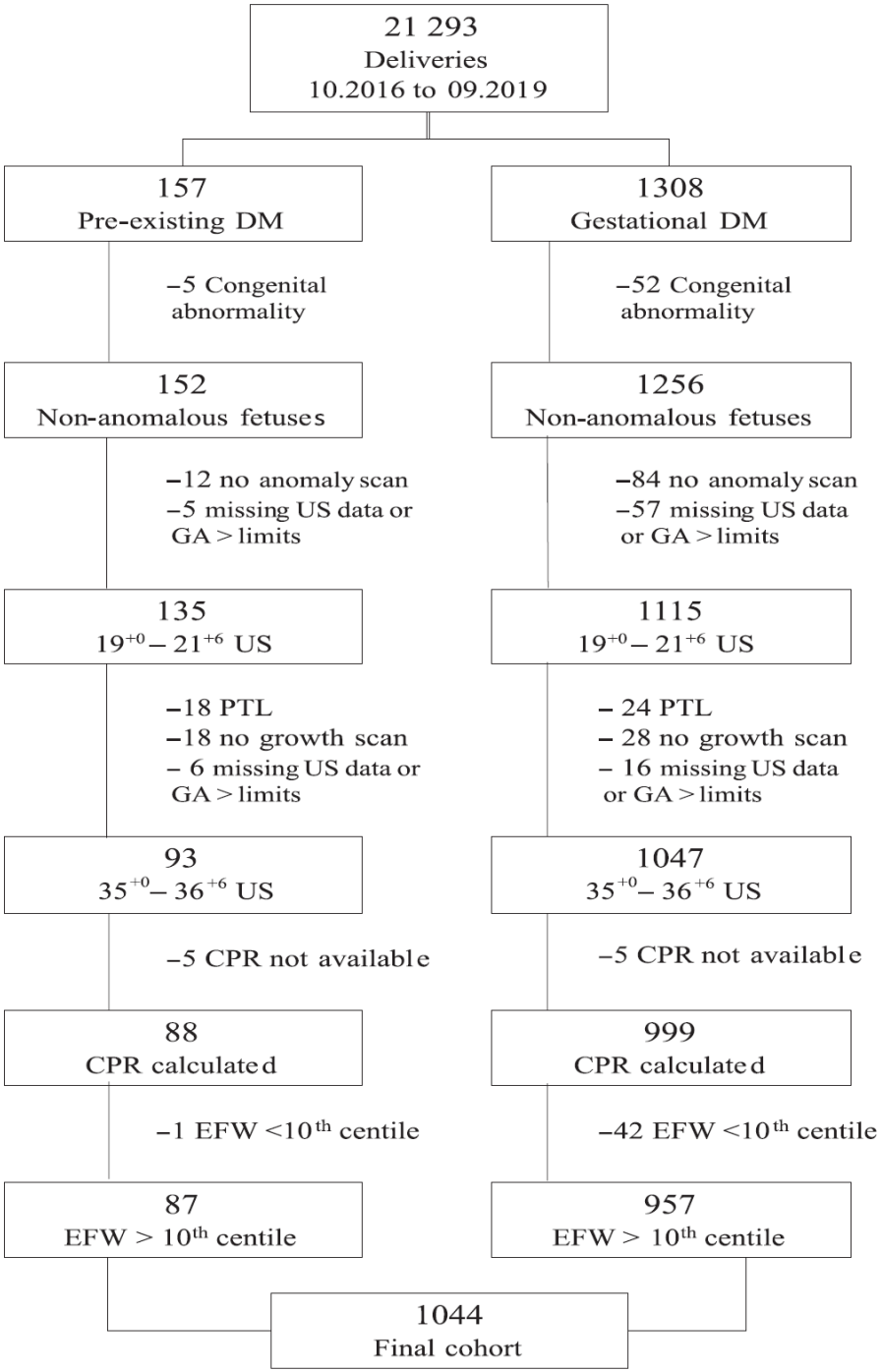


Environmental Science and Pollution Research (2022) 29:19860–19868
https://doi.org/10.1007/s11356-021-17267-y


A
UMBILICAL ARTERY
NORMAL LOW IMPEDANCE
ABNORMAL HIGH IMPEDANCE
ABSENT END DIASTOLIC FLOW
REVERSE END DIASTOLIC FLOW
UMBILICAL ARTERY
UMBILICAL VEIN
B
UTERINE ARTERIES
NORMAL UTERINE ARTERY IMPEDANCE
INCREASED UTERINE ARTERY IMPEDANCE WITH NOTCH
C
DUCTUS VENOSUS
DUCTUS VENOSUS
UMBILICAL VEIN
INFERIOR VENA CAVA
NORMAL DUCTUS VENOSUS
ABSENT A-WAVE
REVERSE A-WAVE
D
MIDDLE CEREBRAL ARTERY
NORMAL HIGH IMPEDANCE MIDDLE CEREBRAL ARTERY
ABNORMAL LOW IMPEDANCE MIDDLE CEREBRAL ARTERY
MIDDLE CEREBRAL ARTERY
CIRCLE OF WILLS
ANTERIOR CEREBRAL ARTERY

Ultrasound pr … in pregnancy complicated b … al diabetes

… A … or more of perinatal … the presence of one 5-minute Apgar less … on to Neonatal Unit, promise. … ection for fetal com-



pondence
Garbagnati, Fetal Medicine Unit, Women's Centre, John Radcliffe al, Headley Way, Oxford, OX3 9DU,

narta.garbagnati@ouh.nhs.uk

Acta Obstet Gynecol Sc

Ultrasound predictors of adverse outcome in pregnancy complicated by pre-existing and gestational diabetes

	All patients	CAO present	CAO absent	
	n = 1044 (100%)	*n* = 174 (16.7%)	870 (83.3)	*p* value
Maternal age (year), mean ± SD	32.4 ± 5.5	31.5 ± 5.3	32.6 ± 5.5	0.018
Body mass index (kg/m^2), mean ± SD	30.4 ± 6.8	31.0 ± 7.1	30.2 ± 6.7	0.169
Body mass index ≥ 30 kg/m^2, *n*/*N* (%)	520/1038 (50.1)	93/173 (53.8)	427/865 (49.4)	0.291
Caucasian ethnicity, *n*/*N* (%)	519/719 (72.2)	71/110 (64.5)	448/609 (73.6)	0.052
Nulliparity, *n* (%)	413 (39.6)	100 (57.5)	313 (36.0)	<0.001
In vitro conception, *n* (%)	23 (2.2)	3 (1.7)	20 (2.3)	0.637
Hypertension, *n*/*N* (%)	65/720 (9.0)	11/109 (10.1)	54/611 (8.8)	0.674
Smoker, *n*/*N* (%)	100/1035 (9.7)	18/173 (10.4)	82/862 (9.5)	0.717
Pre-eclampsia, *n* (%)	68 (6.5)	21 (12.1)	47 (5.4)	0.001
Male fetuses, *n* (%)	562 (53.8)	99 (56.9)	463 (53.2)	0.374
Gestational age at delivery (days), mean ± SD	273.9 ± 8.6	271.0 ± 10.2	274.5 ± 8.1	<0.001
Birth at <37^{+0} weeks, *n* (%)	38 (3.6)	17 (9.8)	21 (2.4)	<0.001
Induction of labor, *n* (%)	481 (46.1)	85 (48.9)	396 (45.5)	0.421

Note: Continuous variables were compared using the Mann-Whitney *U* test for non-normally distributed independent samples.Categorical variables were compared using a chi-squared test. CAO: Composite adverse outcome: 1+ of perinatal death, arterial cord pH < 7.1, admission to neonatal unit, 5-minute Apgar <7, severe hypoglycemia requiring treatment, or CS for suspected fetal compromise.

Acta Obstet Gynecol Scand. 2022;101:787–793.

Ultrasound predictors of adverse outcome in pregnancy complicated by pre-existing and gestational diabetes

TABLE 2 Association between the ultrasound markers estimated fetal weight ≥90th centile, abdominal circumference growth velocity ≥90th centile, cerebro-placental ratio ≤5th centile, and polyhydramnios and the incidence of composite adverse outcome

		Fetuses	CAO present	
		N (%)	*N* (%)	OR (95% CI)
EFW	CPR	1044 (100)	174 (16.7)	
10th–90th	≤5th	64 (6.1)	14 (8.0)	1.44 (0.78–2.66)
	>5th	845 (80.9)	126 (72.4)	1 (reference)
≥90th	≤5th	11 (1.1)	6 (3.4)	6.85 (2.06–22.78)
	>5th	124 (11.8)	28 (16.1)	1.66 (1.05–2.64)

Appropriate for gestational age fetuses — CAO present

All hyper
Total
Risk fa
EFW ≥
ACGV
CPR ≤
Polyhy
GDM on
Total
Risk factor
EFW ≥90th
ACGV ≥90th
CPR ≤5th
Polyhydramni
Pre-existing DM
Risk factor
EFW ≥ 90th
ACGV ≥ 90th
CPR ≤ 5th
Polyhydramni

Note: OR were cal
admission to neon

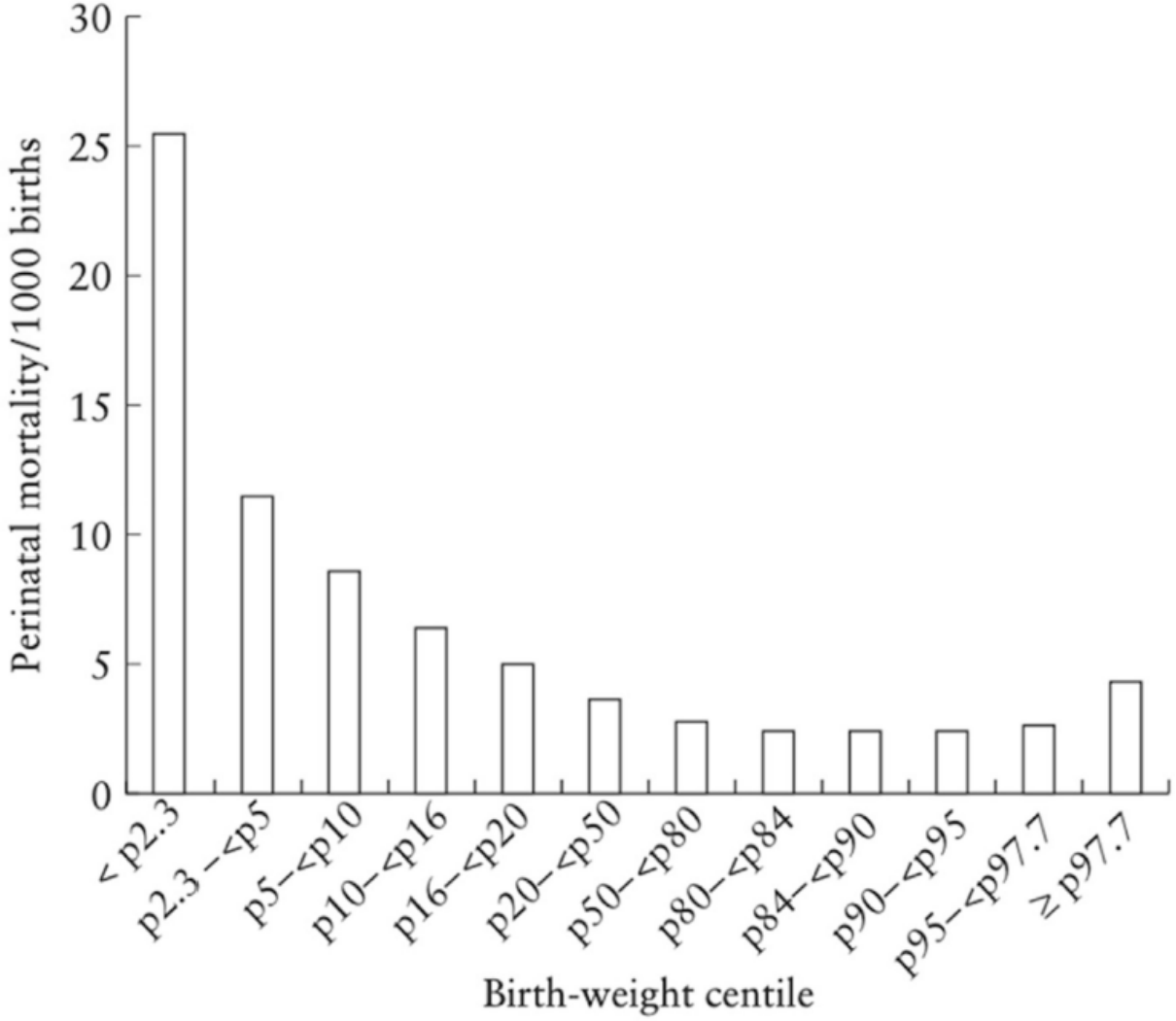
Abbreviations: AC
diabetes mellitus;

[a]Adjusted OR was calculated with logistic regression, including EFW ≥90th centile, ACGV ≥90th centile, CPR ≤5th centile.

Key message

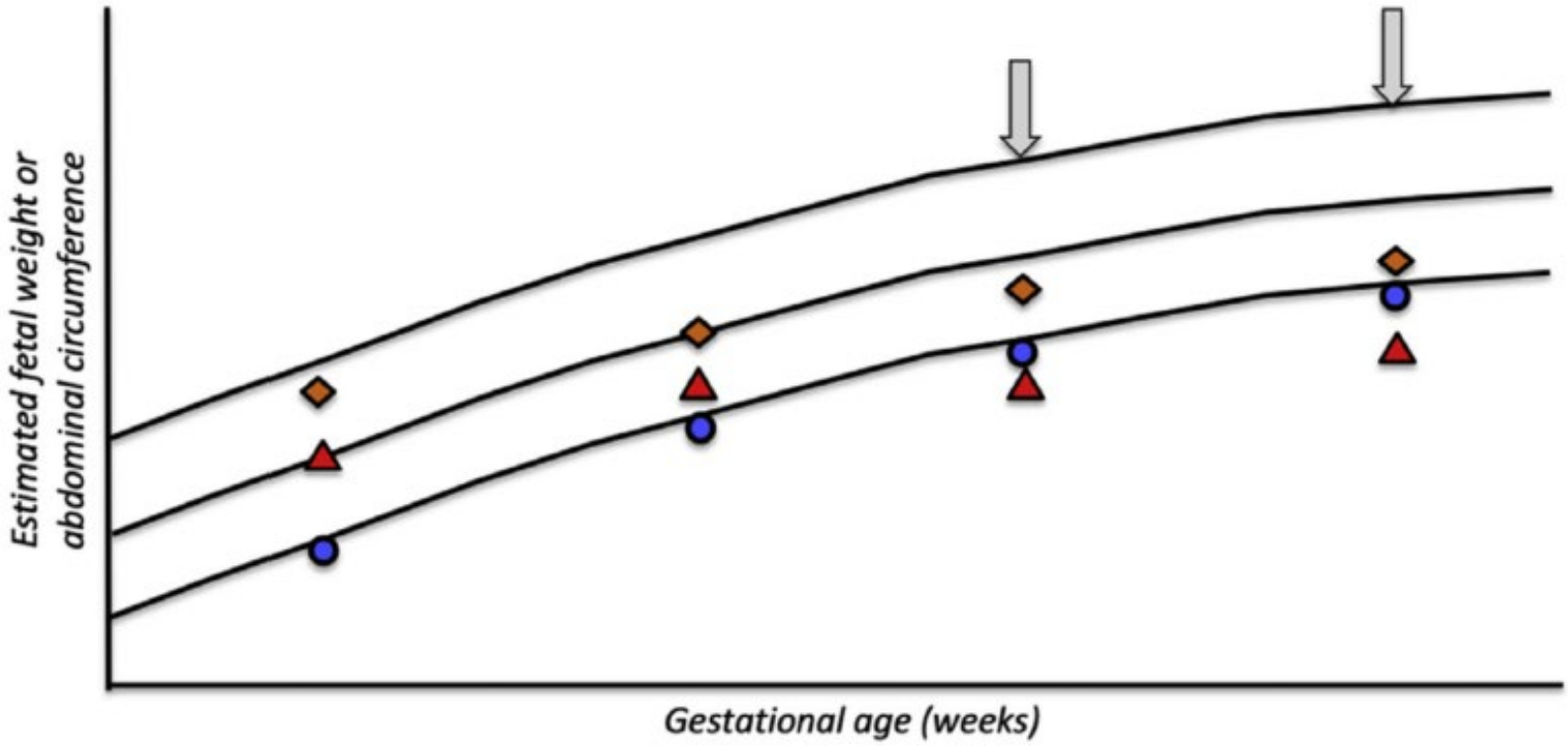
In hyperglycemic pregnancies, large for dates and low cerebro-placental ratio are independent and mutually enhancing risk factors for adverse perinatal outcome. It is the low cerebro-placental ratio and not the abdominal circumference growth acceleration that predicts those large fetuses at most risk.

Clinical Opinion: The diagnosis and management of suspected fetal growth restriction: an evidence-based approach



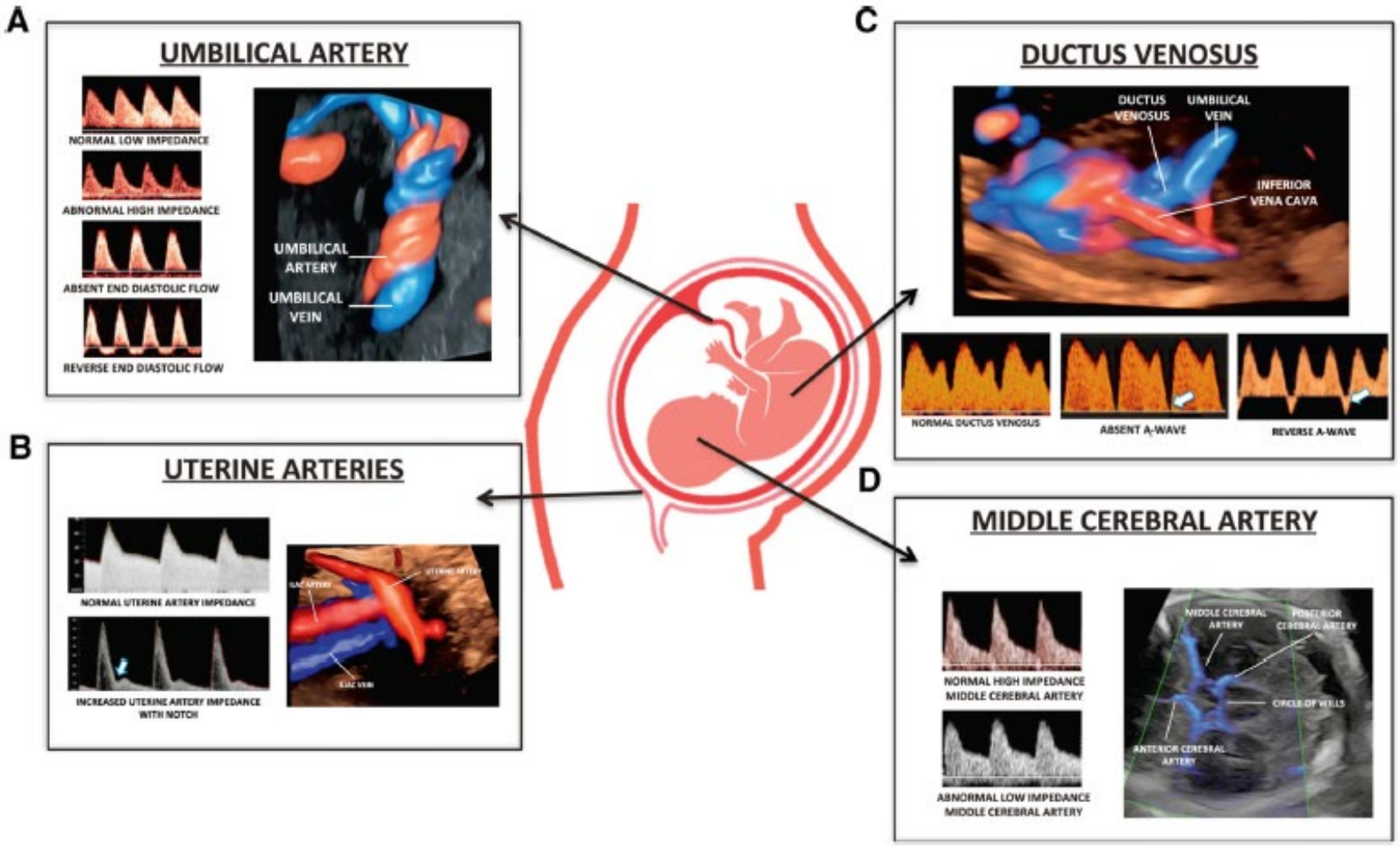
Am J Obstet Gynecol. Author manuscript; available in PMC 2023 March 01.

Clinical Opinion: The diagnosis and management of suspected fetal growth restriction: an evidence-based approach



Am J Obstet Gynecol. Author manuscript; available in PMC 2023 March 01.

Clinical Opinion: The diagnosis and management of suspected fetal growth restriction: an evidence-based approach



Am J Obstet Gynecol. Author manuscript; available in PMC 2023 March 01.

Clinical Opinion: The diagnosis and management of suspected fetal growth restriction: an evidence-based approach

Different phenotypes of fetal growth restriction LATE DOPPLER CHANGES

Evaluation of a single biometric fetal measurement defines the size and not fetal growth. Fetoplacental Doppler and evaluation of fetal growth velocity are important tools to identify uteroplacental insufficiency associated with suspected FGR, allowing its differentiation from a constitutionally small fetus.

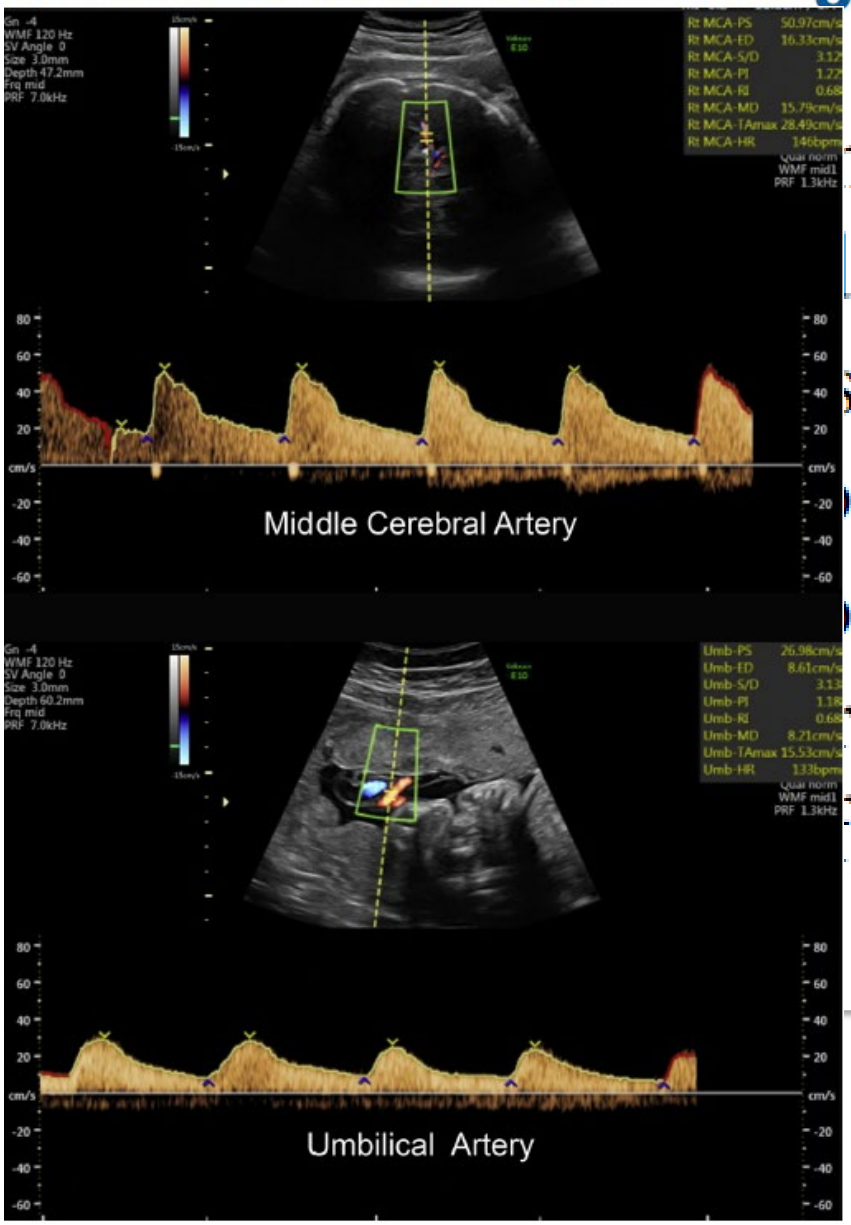
Level 1 evidence supports the application of UA and ductus venosus Doppler in the monitoring of early FGR. High-quality observational studies support the application of the middle cerebral artery and/or its ratios to UA in the surveillance of late suspected FGR. Collectively, these Doppler parameters can predict fetal deterioration and guide optimal surveillance intervals.

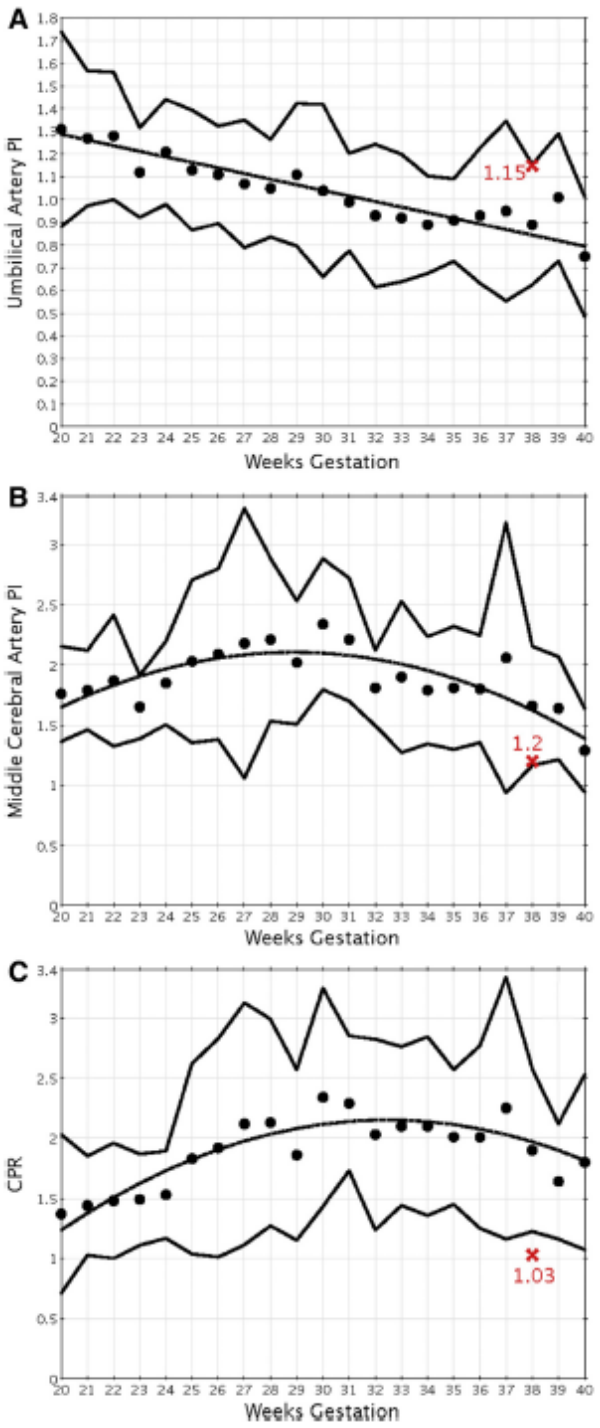
cCTG analysis of fetal heart rate variation and, in particular, the STV provided an objective basis on which to evaluate the fetal condition. Low STV was associated with fetal acidemia and fetal death, and there was strong evidence that monitoring early suspected FGR with ductus venosus Doppler and STV from cardiotocography leads to optimal perinatal outcome.

Am J Obstet Gynecol. Author manuscript; available in PMC 2023 March 01.

The importance of the cerebropla
evaluation of fetal well-being in S
es

JULY 2015 American Journal of Obstetrics & Gynecology





The importance of the cerebroplacental ratio in the evaluation of fetal well-being in SGA and AGA fetuses

JULY 2015 American Journal of Obstetrics & Gynecology

TABLE 4

Sensitivities, specificities, and odds ratio for CPR computations for detecting adverse perinatal outcome[a30]

Cerebroplacental ratio	Measurement standard	Sensitivity	Specificity	Odds ratio
<1	Pulsatility index	66%	85%	11.7
<1	Resistance index	66%	84%	11.8
Less than fifth centile (cross-sectional study)[9]	Centile	80%	60%	6.2
Less than fifth centile (longitudinal study)[11]	Centile	85%	41%	4.1

CPR, abnormal cerebroplacental ratio.

[a] Intraventricular hemorrhage, periventricular leukomalacia, hypoxic ischemic encephalopathy, necrotizing enterocolitis, bronchopulmonary dysplasia, sepsis, and death.

DeVore. Cerebroplacental ratio in fetal well-being in SGA and AGA fetuses. Am J Obstet Gynecol 2015.

The fetal cerebro-placental ratio in diabetic pregnancies is influenced more by the umbilical artery rather than middle cerebral artery pulsatility index

Table 1
Maternal demographics, intrapartum outcomes and ultrasound characteristics stratified by DM type.

Variable	pT1DM (n = 124)	pT2DM (n = 68)	GDM (n = 1089)	p-value
Maternal age (years)§	29.1 [5.2]	33.6 [5.9]	32.5 [5.2]	<0.0001
BMI (kg/m^2)^	25.4 [22.4–29.2]	30.9 [27.6–37.7]	25.9 [22.1–30.8]	<0.0001
Ethnicity*				
Caucasian	109 (87.9)	33 (48.5)	421 (38.7)	<0.0001
Asian	2 (1.6)	9 (13.2)	259 (23.8)	<0.0001†
Indigenous	2 (1.6)	5 (7.4)	6 (0.6)	<0.001†
Indian	0 (0)	4 (5.9)	148 (13.6)	<0.0001†
Other	8 (6.5)	13 (19.1)	236 (21.7)	<0.001
Parity*				
0	60 (48.4)	19 (27.9)	430 (39.5)	0.020
≥1	64 (51.6)	49 (72.1)	659 (60.5)	0.020
ART*	4 (3.2)	0 (0)	65 (6.0)	0.042†
PET/Hypertension*	34 (27.4)	34 (35.3)	105 (9.7)	<0.0001
Thyroid disease*	20 (16.1)	2 (2.9)	95 (8.7)	<0.01†
Smoking*	34 (27.4)	15 (22.1)	126 (11.6)	<0.0001
Total length labour (mins)^	418.5 [250.5–599] (n = 50)	227 [133–313] (n = 38)	276.0 [167–443] (n = 753)	<0.001
Mode of Delivery*				
SVD	22 (17.7)	25 (36.8)	524 (48.1)	<0.0001
Instrumental	10 (8.1)	8 (11.8)	138 (12.7)	0.33
CS	92 (74.2)	35 (51.5)	427 (39.2)	<0.0001
Elective CS	52 (41.9)	16 (23.5)	221 (20.3)	<0.0001
Emergency CS	40 (32.3)	19 (27.9)	206 (18.9)	<0.001
NRFS	8 (6.5)	4 (5.9)	43 (3.9)	0.26†
Other	32 (25.8)	15 (22.1)	163 (15.0)	<0.01
Gestation at US (weeks)§	35.4 [0.8]	35.588 [0.863]	35.633 [0.882]	0.021
CPR§	1.86 [0.60]	1.94 [0.50]	2.08 [0.55]	<0.0001
UA PI§	0.94 [0.21]	0.87 [0.17]	0.85 [0.17]	<0.0001
MCA PI§	1.66 [0.38]	1.63 [0.35]	1.71 [0.34]	0.081
CPR <5th centile*	14 (11.3)	2 (2.9)	38 (3.5)	0.001†
CPR <10th centile*	24 (19.4)	6 (8.8)	87 (8.0)	<0.001
UA PI >90th centile*	23 (18.5)	7 (10.3)	82 (7.5)	<0.001
MCA PI <5th centile*	8 (6.5)	5 (7.4)	44 (4.0)	0.19†

The fetal cerebro-placental ratio in diabetic pregnancies is influenced more by the umbilical artery rather than middle cerebral artery pulsatility index

Table 2
Perinatal outcomes stratified by DM type.

Variable	pT1DM (n = 124)	pT2DM (n = 68)	GDM (n = 1089)	p-value	OR (95% CI) ‡	p-value
Gestation at delivery (weeks) [SD] §	36.7 [1.1]	37.3 [1.0]	38.0 [1.2]	<0.0001	NA	
Delivery <37 weeks (n, %)*	87 (70.2)	31 (45.6)	304 (27.9)	<0.0001	5.77 (3.88–8.74)	<0.0001
BW (g) [SD] §	3515.1 [610.8]	3292.5 [587.0]	3227.8 [535.7]	<0.0001	NA	
BW <10th centile (n, %)*	4 (3.2)	3 (4.4)	55 (5.1)	0.72†	0.19 (0.051–0.52)	0.003
LGA (BW ≥ 90th centile) (n, %)*	33 (26.6)	13 (19.1)	111 (10.2)	<0.0001	3.82 (2.34k6.15)	<0.0001
Apgar score <7 at 5 min (n, %)*	3 (2.4)	2 (2.9)	21 (1.9)	0.63†	0.55 (0.12–1.84)	0.38
Acidosis (pH ≤ <7.0 or Lactate>6) (n, %)*	11 (8.9)	4 (5.9)	68 (6.2)	0.47†	1.81 (0.83–3.66)	0.11
NCCU admission (n, %)*	66 (53.2)	14 (20.6)	186 (17.1)	<0.0001	2.91 (1.85–4.57)	<0.0001
Respiratory distress (n, %)*	40 (32.3)	9 (13.2)	160 (14.7)	<0.0001	1.43 (0.89–2.25)	0.13
Hypoglycaemia (n, %)*	52 (43.0)	13 (19.4)	129 (11.9)	<0.0001	2.59 (1.64–4.04)	<0.0001
Perinatal Death (n, %)*	0 (0)	0 (0)	5 (0.5)	NA	NA	NA
Composite adverse neonatal outcome (n, %)*	91 (74.6)	27 (39.7)	349 (32.2)	<0.0001	3.39 (2.16–5.42)	<0.0001

§ Mean (SD)—data analysed by 1-way ANOVA.
*Number (percentage)—data analysed by Chi-squared test or Fisher's exact test where indicated (†).
‡ Odds ratios are for the Insulin-treated group compared to other treatment groups.
BW—birth weight, LGA—large for gestational age, NCCU—neonatal intensive care unit, SD—standard deviation, OR—Odds Ratio, 95% CI—95% Confidence Interval.
OR adjusted for birthweight and gestational age at delivery with exception of delivery <37 weeks which is unadjusted and birth weight <10th centile and LGA which are adjusted for gestational age only.

Improved neonatal outcomes in pregnancies with coexisting gestational diabetes and preeclampsia in normal birthweight neonates- insights from a retrospective cohort study

Introduction: We aimed to assess neonatal and maternal outcomes in appropriate-for-gestational-weight (AGA) neonates of mothers with both gestational diabetes mellitus (GDM) and preeclampsia (PET).

Methods: Medical records of women diagnosed with GDM or PET were reviewed. Women with AGA neonates were divided into three groups- GDM, PET, and GDM + PET and maternal neonatal and placental outcomes were compared. The primary outcome was a composite of adverse neonatal outcomes, including intensive care unit admission (NICU), neurological morbidity, hypoglycemia, ventilation, respiratory distress syndrome (RDS), phototherapy, sepsis, blood transfusion, and neonatal death. Post-hoc analysis was performed to determine between-group significance.

Results: Composite adverse neonatal outcomes are significantly lower in women with multiple morbidities compared to women with confined PET ($p = 0.015$), and a similar trend is observed when comparing neonatal outcomes between women with GDM to those with GDM + PET, yet these results are underpowered (18.9 % vs. 12.8 % respectively, $p = 0.243$). Placentas of women with GDM + PET were larger, with a lower rate of placentas below the 10th percentile as compared to placentas of women with isolated PET ($p < 0.001$), but with similar rates of MVM lesions.

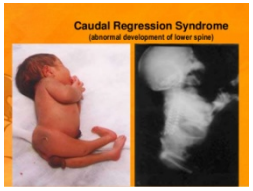
Discussion: While maternal and placental outcomes in patients of the GDM + PET group resemble the characteristics of the PET group, surprisingly, the neonatal outcomes in this group are significantly better compared to isolated morbidities. The paradoxical benefit attributed to the coexistence of GDM + PET may be explained by a balance of the opposing trends characterizing these morbidities-the reduced blood and nutrient supply characterizing PET vs. chronic overflow and abundance typical of GDM.

Clinical trial registration: approval of local ethics committee WOMC-19-0152.

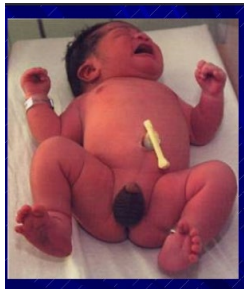
https://doi.org/10.1016/j.placenta.2024.02.008

Malformazioni fetali

Apparato	Malformazioni
Sistema nervoso centrale	Difetti del tubo neurale, oloprosencefalia, agenesia del corpo calloso, sindrome di Arnold Chiari, schizencefalia, agenesia del tratto olfattorio, idrocefalo
cardio vascolare	Trasposizione dei grossi vasi, tetralogia di Fallot, trasposizione dei grossi vasi, arteria ombelicale unica, ipoplasia del cuore sinistro, DIV, DIA, cardiomegalia
gastrointestinale	Stenosi del piloro, atresia duodenale, atresia anorettale, cisti/fistola onfalo-enterica, microcolon
urogenitale	Agenesia renale, idronefrosi, doppio uretere, ureterocele, agenesia uterina, ipospadia, testicoli ipoplasici, criptorchidismo
Muscolo scheletrico	Craniosinostosi, anomalie costovertebrali, palatoschisi, sindrome della regressione caudale, polisindattilia, piede torto
Altri	Microftalmia, coloboma dell'iride, ernia diaframmatica, atresiea delle coane



Balsells M, Garcia-Patterson A, Gich I. Major congenital malformations in women with gestational diabetes mellitus: a systematic review and meta-analysis. Diabetes Metab Res Rev. 2012;28:252–257.



Traumi da parto e asfissia perinatale

distocia di spalla → traumi da parto asfissia

macrosomia → traumi da parto asfissia

traumi da parto asfissia → DANNO NEUROLOGICO

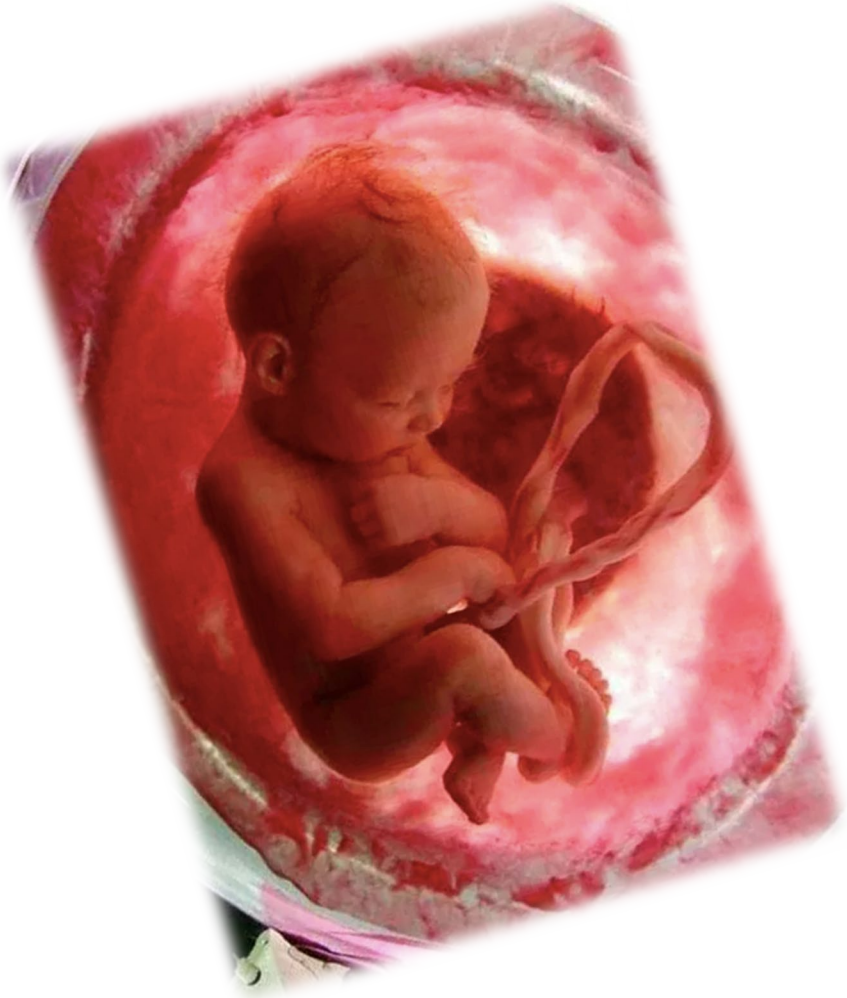
- frattura di clavicola
- frattura di omero
- lesione del plesso brachiale *(0.2% pn<4 Kg vs 3% pn>4 Kg - Gherman et al 2006)*
- lesione del nervo faciale
- traumi addominali
- emorragie oculari

→ **taglio cesareo (35.23 % GDM vs 26.89% non GDM - *Sven et al 2010*)**

Paralisi di Erb 0.42 per mille nati popolazione generale
4.5 per mille nati Studio C.E.M.A.C.H.
Fratture omero o clavicola 8/1124 nati

Esakoff TF, Cheng YW, Sparks TN, Caughey AB. The association between birthweight 4000 g or greater and perinatal outcomes in patients with and without gestational diabetes. Am J Obstet Gynecol. 2009;200:672. e1–672. e4.

Diabete & IUGR



Vasculopatia
diabetica

↓

Ispessimento membrana villi coriali e
sclerosi vasi pelvici,
delle arterie uterine ed ovariche

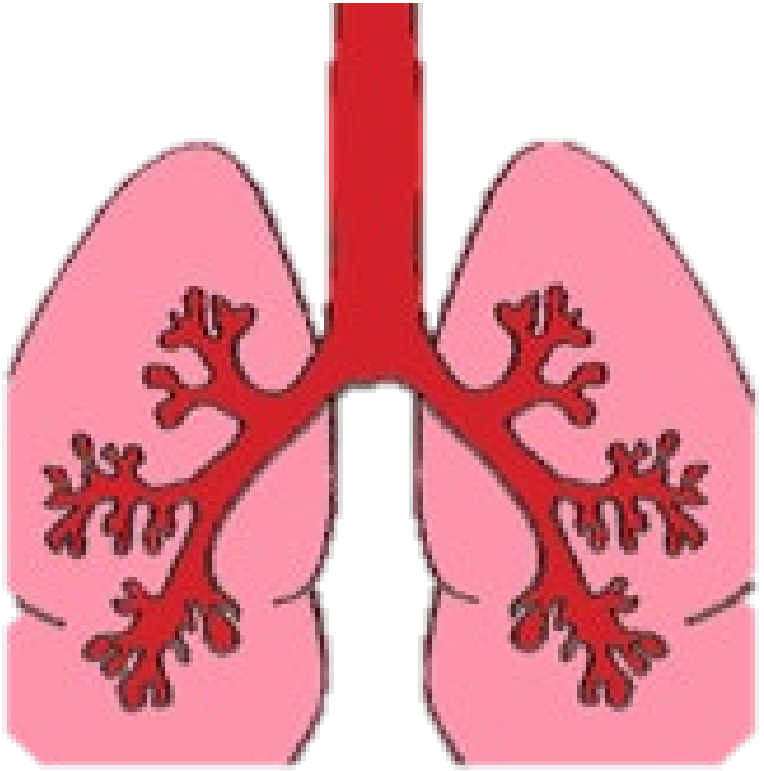
↓

Ipoafflusso di sangue al feto
con IPOSSIA CRONICA

andrea.dotta@opbg.net

Diabete & distress respiratorio

- Macrosomia
- Taglio Cesareo
- Iperglicemia ed iperinsulinismo che determinano deficit surfattante ed immaturità polmonare



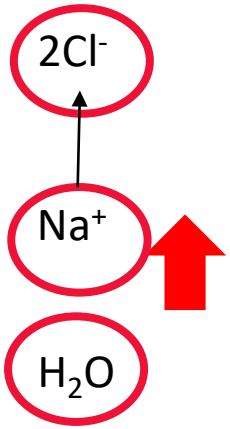
DISTRESS RESPIRATORIO
TACHIPNEA TRANSITORIA

andrea.dotta@opbg.net

Shoar Z. et al 2016 J Obes Weight Loss Ther

Fetal lung fluid balance

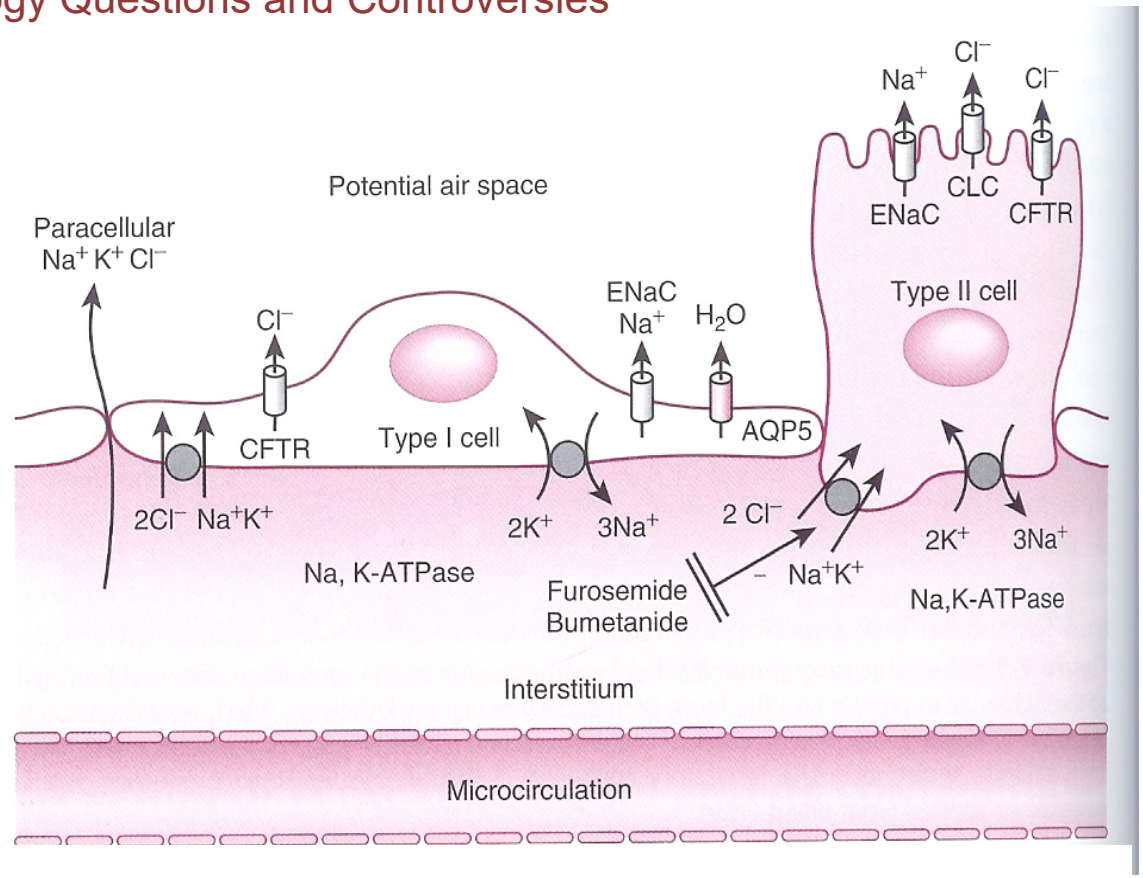
The Newborn Lung- Neonatology Questions and Controversies
Eduardo Bancalari, Ed 2008



Air space

Interstitium

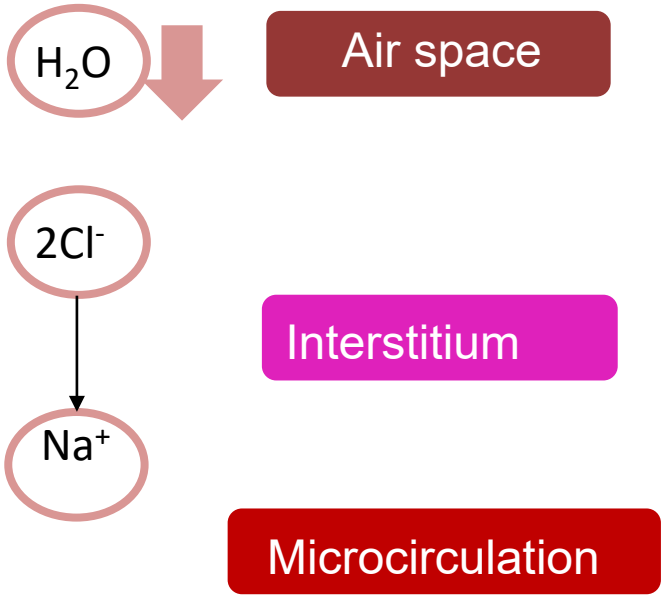
Microcirculation

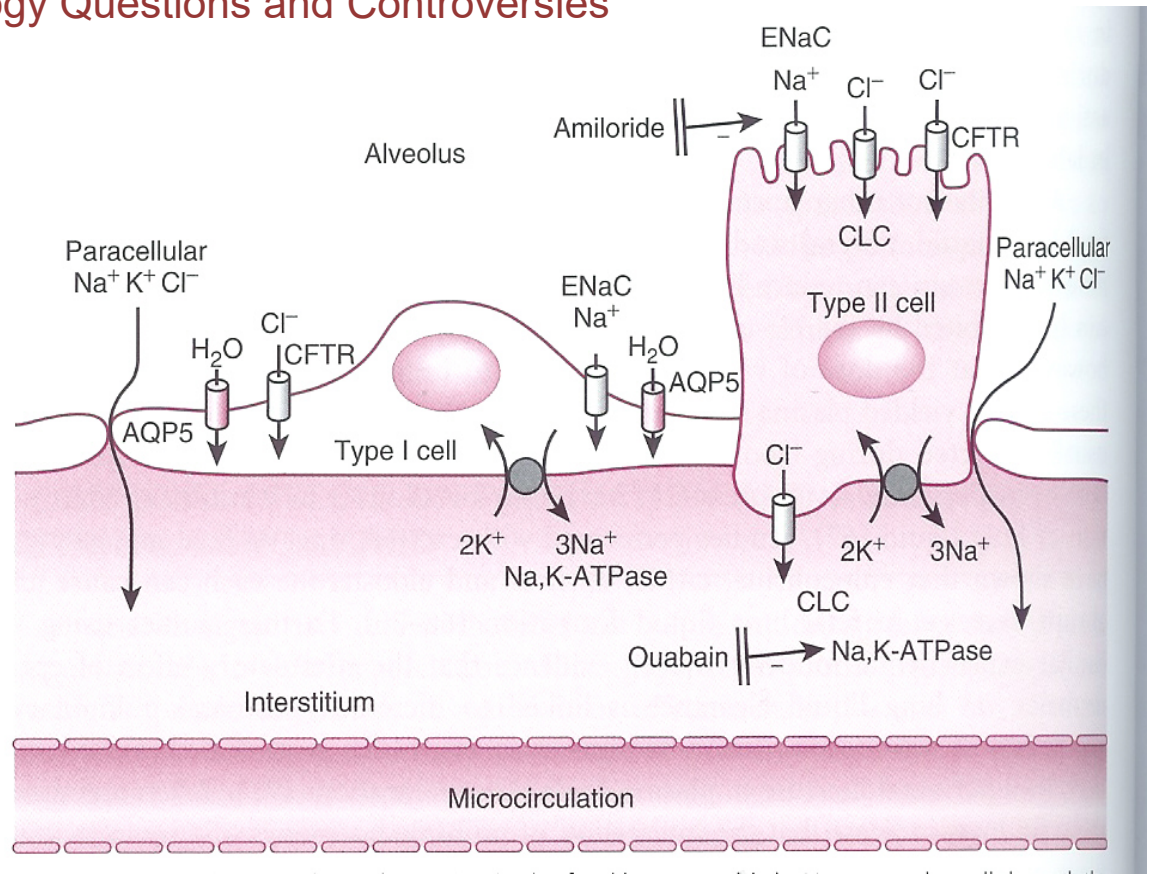


andrea.dotta@opbg.net

Perinatal lung fluid balance

The Newborn Lung- Neonatology Questions and Controversies
Eduardo Bancalari, Ed 2008





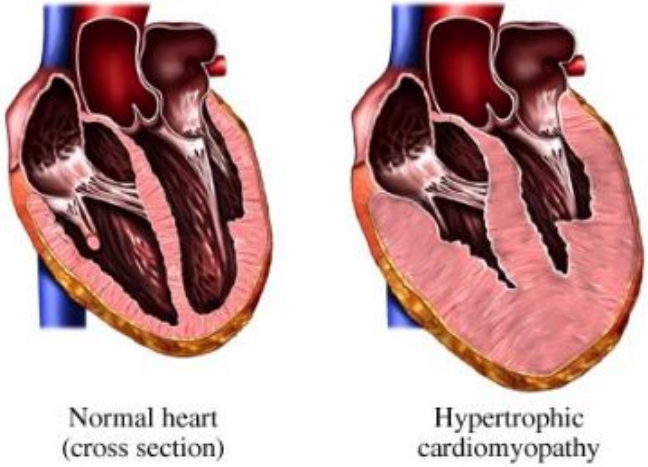
andrea.dotta@opbg.net

Diabete & Cuore

- Livelli **elevati di glicemia non controllata** determinano l'aumento dell'insulina che determina **l'aumento dell'espressione fetale** dei **recettori cardiaci per l'insulina**
- L'insulina, ormone anabolizzante, determina **iperplasia ed ipertrofia del miocardio fetale**, in particolare del setto interventricolare che può condurre a una cardiomiopatia ipertrofica

Sherif F. Elmekkawi et al. 2015 Clinical Medicine Insights: Women's health

Diabete & Cuore



Normal heart (cross section)

Hypertrophic cardiomyopathy

Incidenza di cardiopatie congenite > 5 volte rispetto alla popolazione generale
(6% vs 0,8%)

- 3-6% dei figli di madre diabetica sviluppano cardiopatie e il **40% di questi sviluppa una cardiomiopatia ipertrofica**

Sherif F. Elmekkawi et al. 2015 Clinical Medicine Insights: Women's health

L'importanza del controllo glicemico

Table 1. Comparison between good and poor glycemic control on prenatal and postnatal ultrasound measurements.

	GLYCEMIC CONTROL (110)		*P* VALUE
	GOOD (78)	POOR (32)	
Interventricular septum (mm)			
by prenatal ultrasound	3.78 ± 0.78	5.09 ± 0.96	<0.0001
by postnatal ultrasound	3.81 ± 0.78	5.20 ± 0.93	<0.0001
Right myocardial thickness (mm)			
by prenatal ultrasound	4.36 ± 0.58	5.25 ± 0.76	<0.0001
by postnatal ultrasound	4.38 ± 0.59	5.25 ± 0.76	<0.0001
Left myocardial thickness (mm)			
by prenatal ultrasound	4.97 ± 0.62	5.66 ± 0.75	<0.0001
by postnatal ultrasound	5.01 ± 0.63	5.78 ± 0.81	<0.0001
Ejection fraction (%)			
by postnatal echocardiography	61.79 ± 3.17	57.50 ± 4.02	<0.0001

Sherif Elmekkawi et al. 2015

Ecocardiografia & diabete non controllato

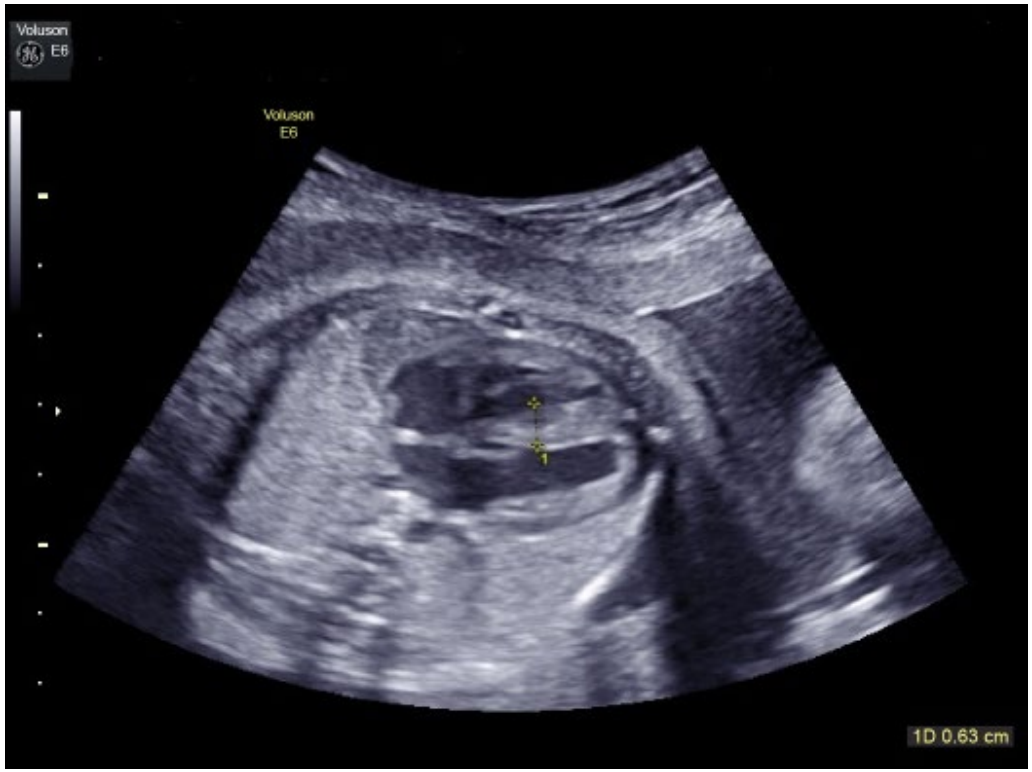


Figure 3. Fetal interventricular septal thickness 6 mm in a case of uncontrolled pregestational diabetes mellitus with pregnancy by 2D ultrasound.

Sherif F. Elmekkawi et al. 2015

andrea.dotta@opbg.net

Diabete e metabolismo

- Subito dopo la nascita si interrompe il passaggio di glucosio dalla madre al feto e ciò determina un **iperinsulinismo** nel neonato

- L'iperinsulisimo determina **un'inibizione della glicogenolisi, gluconeogenesi e lipolisi**

- L'iperisulinismo fetale può **persistere da 1 a 2 ore dopo la nascita** causando quindi ipoglicemia

andrea.dotta@opbg.net

Ipoglicemia neonatale

- Si definisce ipoglicemia **valori ≤ 40mg/dl**
- L'esposizione a valori bassi di glicemia per periodi prolungati si associa a **danni neurologici e alterazioni RMN**
- E' difficile stabilire il livello di glicemia al di sotto del quale si possono manifestare esiti neurologici a breve e lungo termine nel neonato (Mitanchez 2010)
- Un **cattivo controllo glicemico nel terzo trimestre** di gravidanza (HbA1 elevata) e durante il travaglio aumenta il **rischio di ipoglicemia neonatale** *(NCCWCHD 2008, Mikkelsen 2011*)

andrea.dotta@opbg.net

JAMA Pediatrics | Original Investigation

Infants Eligible for Neonatal Hypoglycemia Screening
A Systematic Review

Michelle O'Brien, MBChB; Catherine Gilchrist, PhD; Lynn Sadler, MBChB; Joanne E. Hegarty, PhD; Jane M. Alsweiler, PhD

IMPORTANCE Neonatal hypoglycemia is common, occurring in up to 50% of infants at risk for hypoglycemia (infant of diabetic mother [IDM], small for gestational age [SGA], large for gestational age [LGA], and preterm) and is associated with long-term neurodevelopmental impairment. Guidelines recommend screening infants at risk of hypoglycemia. The proportion of infants who require screening for neonatal hypoglycemia is unknown.

Key Points

Question What is the proportion of infants eligible for neonatal hypoglycemia screening?

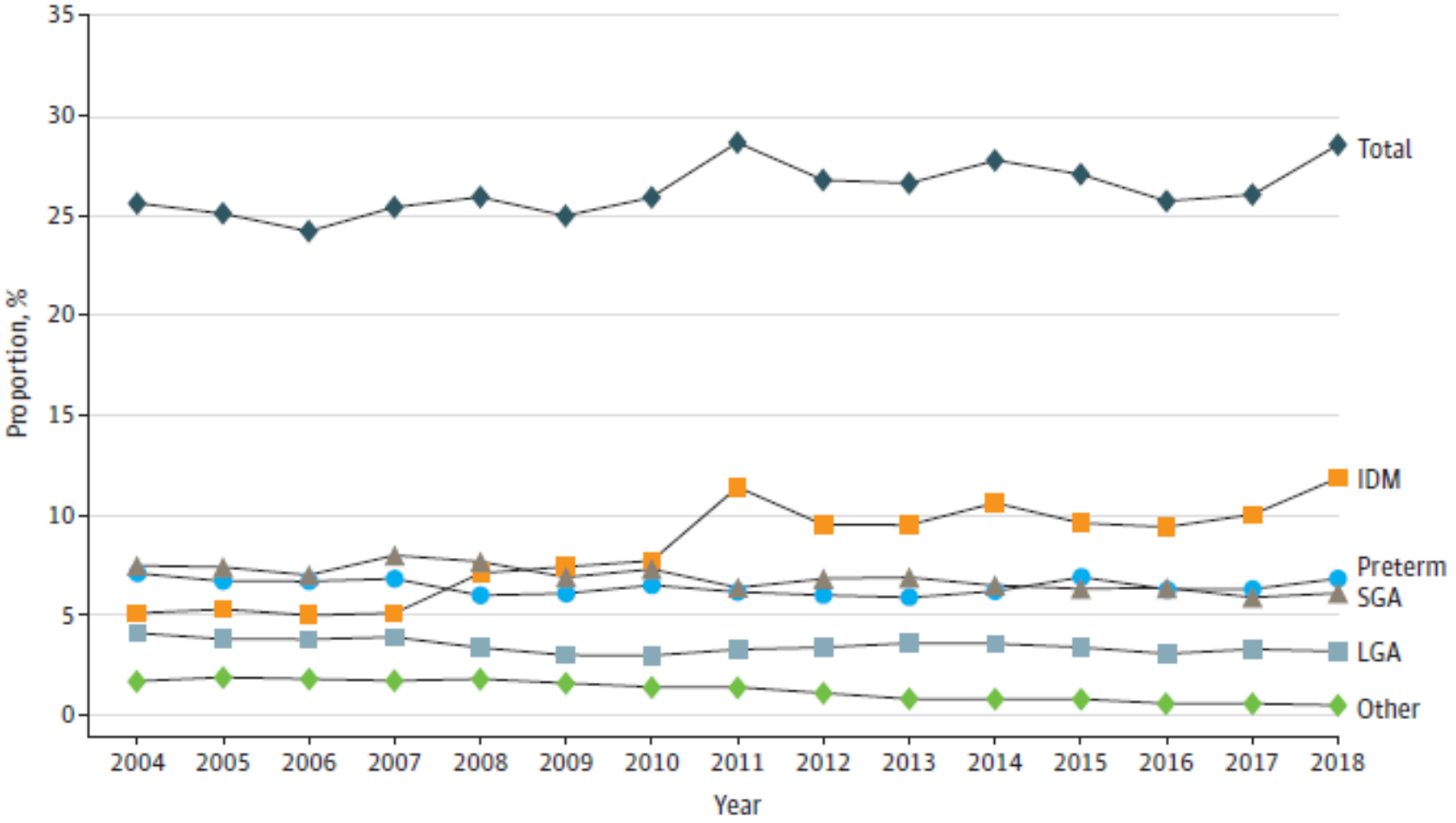
Findings In this review of 18 clinical practice guidelines for neonatal hypoglycemia screening, the Queensland Clinical Guideline: Hypoglycaemia–newborn was identified as the highest-scoring guideline. In this 15-year cohort study of 101 372 eligible infants, the proportion of infants eligible for hypoglycemia screening was 26.3%.

Meaning One-quarter of well infants are eligible for hypoglycemia screening.

JAMA Pediatr. 2023;177(11):1187-1196. doi:10.1001/jamapediatrics.2023.3957
Published online October 2, 2023.

JAMA Pediatr. 2023;177(11):1187-1196. doi:10.1001/jamapediatrics.2023.3957
Published online October 2, 2023.

Figure 2. Eligibility of Infants Born in Auckland City Hospital for Neonatal Hypoglycemia Screening



Published in a peer-reviewed journal or by international, national, or statewide clinical representative groups

IDM indicates infant of diabetic mother; SGA, small for gestational age; LGA, large for gestational age.

Oral dextrose gel to prevent hypoglycaemia in at-risk neonates (Review)

Summary of findings 1. Dextrose gel compared with placebo for prevention of hypoglycaemia in newborn infants

Dextrose gel compared with placebo for prevention of hypoglycaemia in newborn infants					
Patient or population: newborn infants at risk of neonatal hypoglycaemia **Setting:** New Zealand and Australia **Intervention:** dextrose gel **Comparison:** placebo gel					
Outcomes	**Anticipated absolute effects***		**Relative effect**	**No. of partici-**	**Certainty of**	**Comments**
(investigator-defined, any treatment - oral dextrose gel, intravenous dextrose, or other drug therapy) during initial hospital stay (yes/no)						
Receipt of intravenous treatment for hypoglycaemia (yes/no)	37 per 1000	37 per 1000 (25 to 55)	RR 1.01 (0.68 to 1.49)	2548 (2 RCTs)	⊕⊕⊕⊖ MODERATE[a]	
Adverse events# (e.g. choking or vomiting at time of administration)	10 per 1000	12 per 1000 (6 to 24)	RR 1.22 (0.64 to 2.33)	2510 (2 RCTs)	⊕⊕⊕⊖ MODERATE[a]	

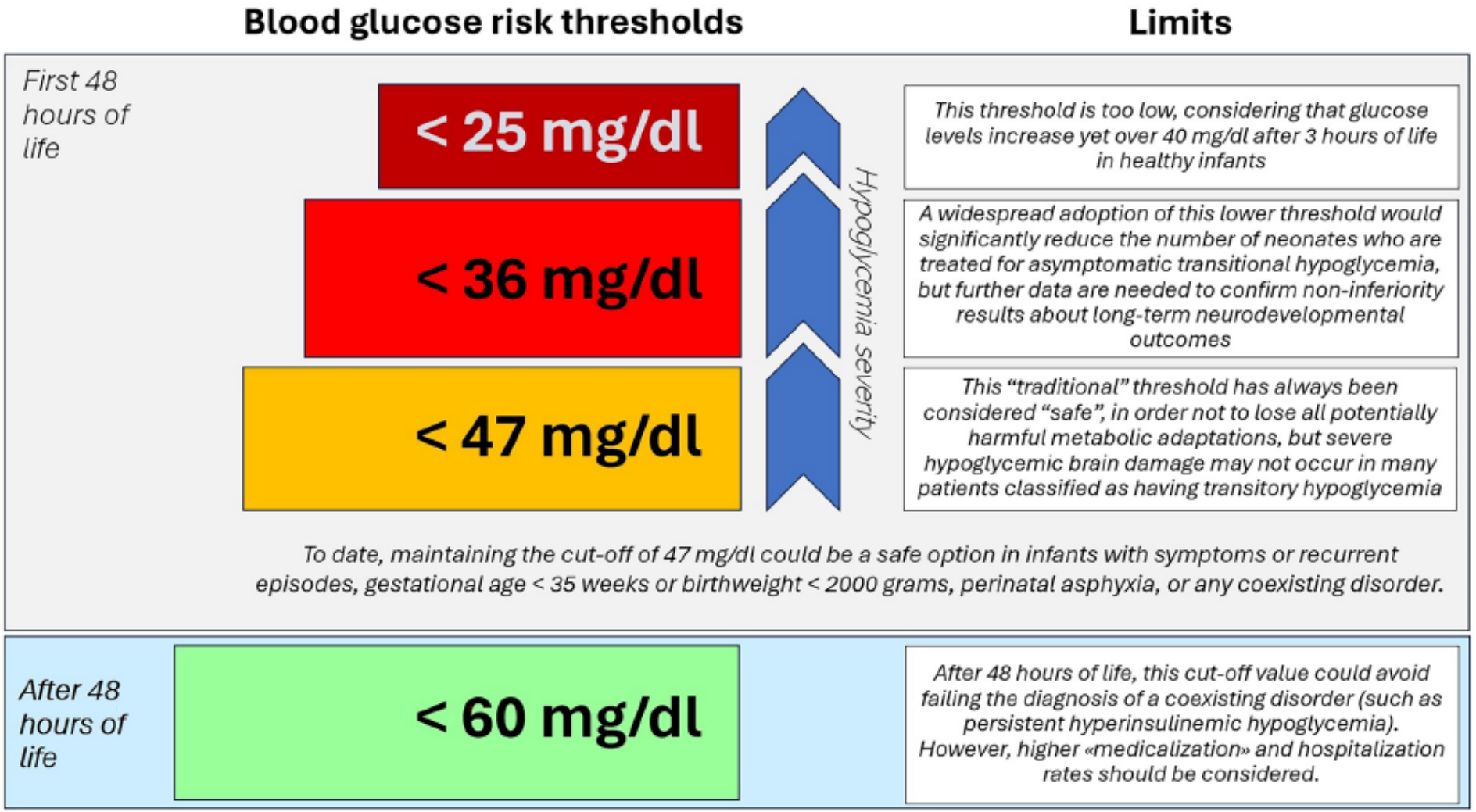
Authors' conclusions

Prophylactic oral dextrose gel reduces the risk of neonatal hypoglycaemia in at-risk infants and probably reduces the risk of treatment for hypoglycaemia without adverse effects. It may make little to no difference to the risk of major neurological disability at two years, but the confidence intervals include the possibility of substantial benefit or harm. Evidence at six to seven years is limited to a single small study.

In view of its limited short-term benefits, prophylactic oral dextrose gel should not be incorporated into routine practice until additional information is available about the balance of risks and harms for later neurological disability. Additional large follow-up studies at two years of age or older are required. Future research should also be undertaken in other high-income countries, low- and middle-income countries, preterm infants, using other dextrose gel preparations, and using comparators other than placebo gel. There are three studies awaiting classification and one ongoing study which may alter the conclusions of the review when published.

Roberts L, Lin L, Alsweiler J, Edwards T, Liu G, Harding JE.
Oral dextrose gel to prevent hypoglycaemia in at-risk neonates.
Cochrane Database of Systematic Reviews 2023, Issue 11. Art. No.: CD012152.
DOI: 10.1002/14651858.CD012152.pub4.

Neonatal hypoglycemia and neurodevelopmental outcomes: Yesterday, today, tomorrow



Domenico Umberto De Rose[1] · Alessandro Perri[2] · Luca Maggio[3,4] · Guglielmo Salvatori[1] · Andrea Dotta[1] · Giovanni Vento[2,3] · Francesca Gallini[3,5]

Neonatal hypoglycemia and neurodevelopmental outcomes: Yesterday, today, tomorrow

Table 3 Our simplified evidence-based algorithm for the management of neonatal hypoglycemia in the first 48 h of life

Blood glucose (mg/dl)	Other factors	Oral dextrose gel followed by milk administration	Dextrose intravenous "mini-bolus"	Dextrose continuous intravenous infusion
< 36	-	✗ *(during placement of the venous access if not already available)*	(✗)	(✗)
36–47	No	✗		
36–47	Symptoms or coexisting disorders (e.g., perinatal asphyxia)	✗ *(during placement of the venous access if not already available)*	✗	✗
36–47	Late prematurity or low birth weight	✗ *(during placement of the venous access if not already available)*		✗
36–47	Low glucose levels although two previous doses of oral dextrose gel			✗

Maternal diabetes (regardless of type or treatment used during pregnancy)

Preeclampsia and pregnancy-induced hypertension (PIH)

Premature birth (< 37 weeks)

Post-term birth (> 42 weeks)

What is Known:

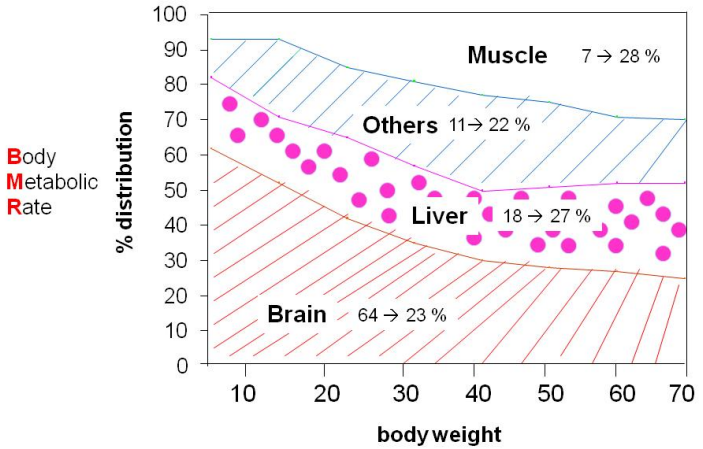
- *Neonatal hypoglycemia (NH) is a well-known cause of brain injury that could be prevented to avoid neurodevelopmental impairment.*
- *Diagnosis is challenging, considering the different suggested operational thresholds for NH (<36, <40, <45, <47 or <50 mg/dl).*

What is New:

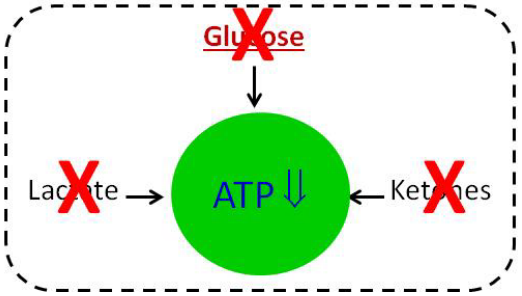
- *A 36 mg/dl threshold seems to be not associated with a worse psychomotor development at 18 months of life when compared to the "traditional" threshold (47 mg/dl).*
- *Further studies on long-term neurodevelopmental outcomes are required before suggesting a more permissive management of NH.*

Twins with discordant weight (> 10%)

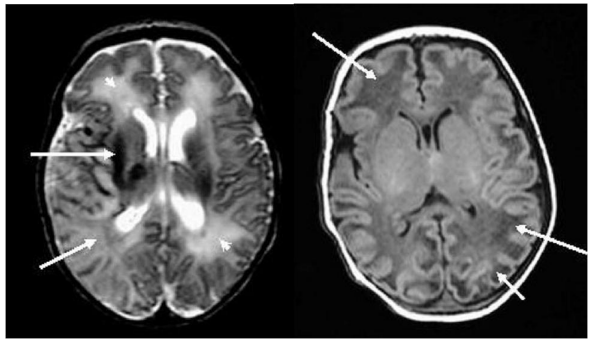
- In the first months of life, plasma glucose concentration provides 90% of cerebral energy
- Building a brain is more expensive than maintaining it: infant's brain consumes 40-60% of the body's basal metabolic rate (20% in adults)



- Brain can use ketones and lactate as alternative energy fuel when their concentration is sufficiently elevated; insulin suppresses ketones and lactate



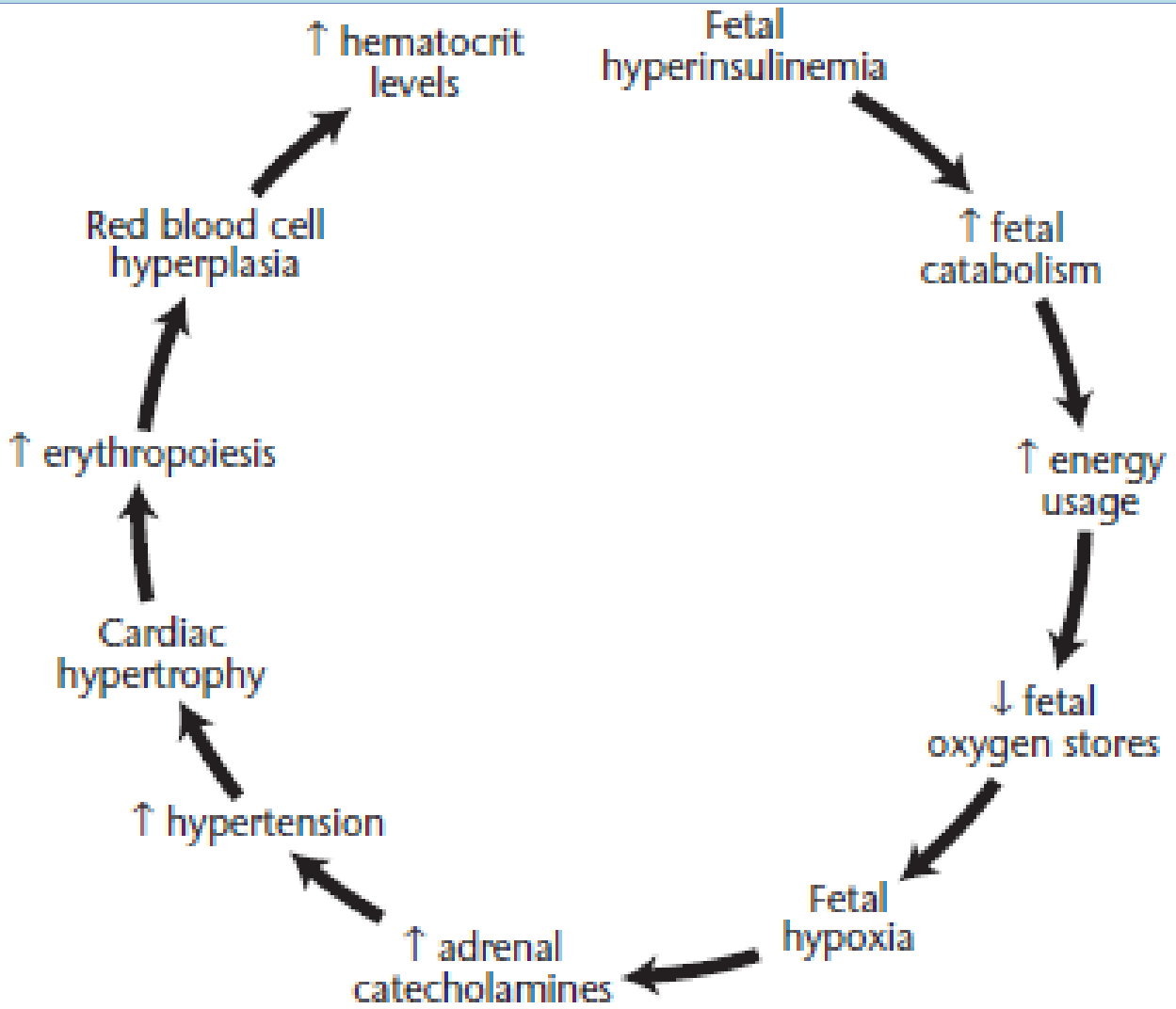
- Developing brain is more susceptible to metabolic insult
- Topography of lesions might be determined by age, consistent with physiological brain maturation (neonates -> posterior white matter; infants -> basal ganglia; children -> parieto-temporal cortex) (Gataullina et al Dev Med Child Neurol 2013)



✓ Factors to consider: age at onset, delay in diagnosis, severity, frequency and duration of hypoglycemia, type of hyperinsulinism, co-morbidities, seizures

Policitemia

Ipos si



andrea.dotta@opbg.net

Policitemia

- La policitemia determina **Hb>20g/dl e Ht >65%**
- E’ asintomatica ma può determinare **infarti cerebrali, NEC, trombosi vena renale**
- L’aumentata distruzione dei GR può comportare **iperbilirubinemia** (ittero)

andrea.dotta@opbg.net

Ipossia cronica

aumentata eritropoiesi

policitemia

traumi da parto

(es. cefaloematoma)

immaturità epatica

(ridotta coniugazione bilirubina)

iperbilirubinemia indiretta

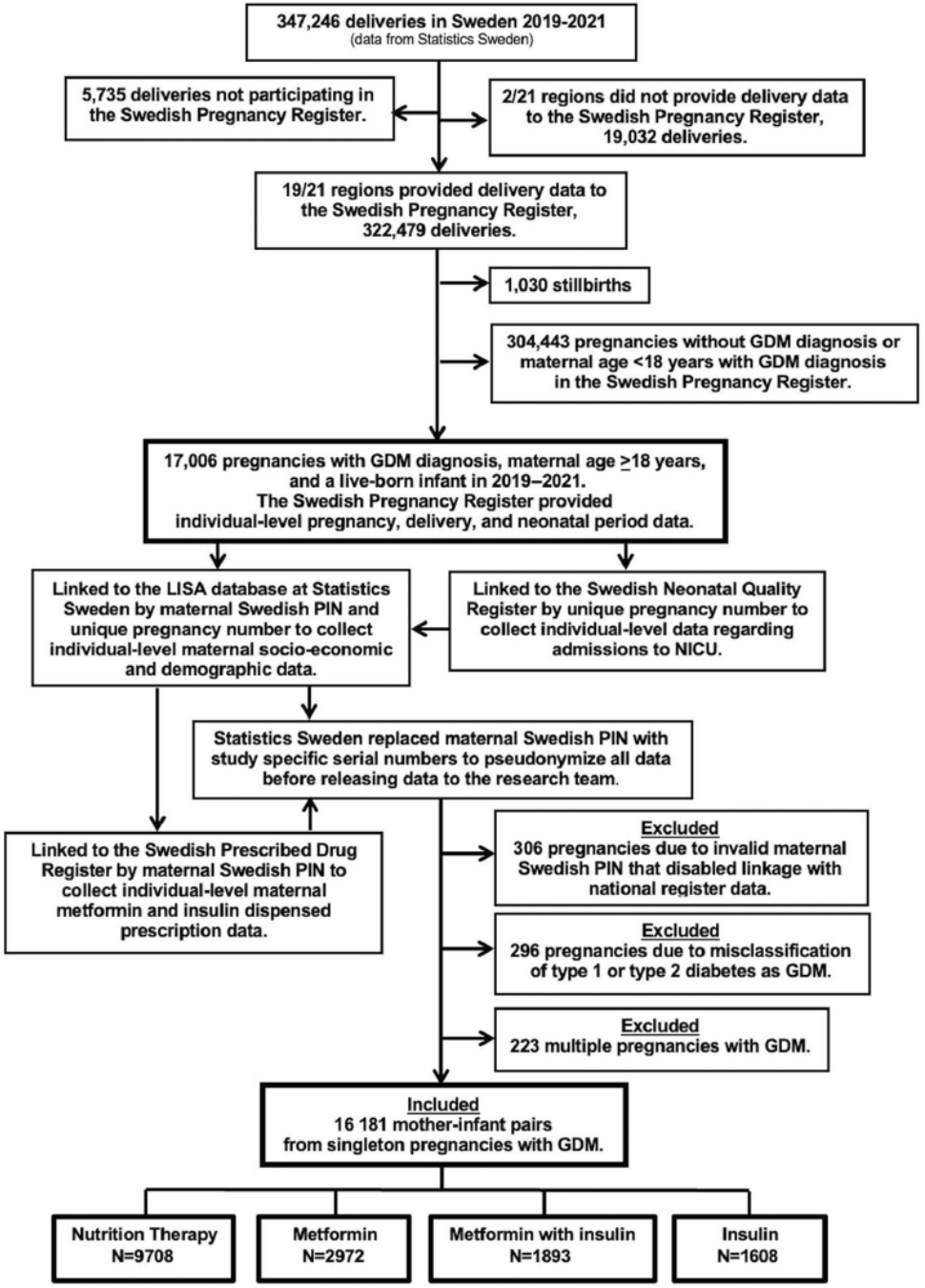
Mitanchez D, Yzydorczyk C, Siddeek B, Boubred F, Benahmed M, Simeoni U. The offspring of the diabetic mother – short- and long-term implications. *Best Pract Res Clin Obstet Gynaecol*. Epub August 20, 2014.

Neonatal out … l gestational diabetes mell … udy

Johanna Molin[1] … | Eszter Vanky[3,4]
Itay Zamir[1] | Eva …

…col Scand. 2024;00:1–16.

Introduction: … iated with gestational diabet… maternal treatment. We aime… spring exposed to metformin t… omparison with nutrition thera… tion, we investigated MT in … eonatal anthropometrics, resp… re, and preterm birth.



	Metformin alone versus metformin combined with insulin (reference)	Metformin alone versus insulin alone (reference)	Metformin combined with insulin versus insulin alone (reference)
	OR (95% CI)	OR (95% CI)	OR (95% CI)
Primary outcome			
Neonatal hypoglycemia[a]			
Crude	0.72 (0.63–0.83)	0.49 (0.43–0.56)	0.68 (0.59–0.79)
Adjusted model[b]	0.74 (0.64–0.87)	0.47 (0.40–0.55)	0.63 (0.53–0.75)
Seco			
LG			
SG			
Re			
Hy			
Ap			
Ap			
Crude	1.28 (0.38–4.24)	1.44 (0.38–5.45)	1.13 (0.25–5.06)
Adjusted model[b]	1.41 (0.41–4.85)	1.41 (0.35–5.80)	1.01 (0.21–4.84)
Preterm birth[e]			
Crude	0.84 (0.67–1.04)	0.87 (0.68–1.10)	1.04 (0.81–1.33)
Adjusted model[b]	0.93 (0.73–1.17)	0.94 (0.71–1.24)	1.02 (0.76–1.35)
NICU			
Crude	0.86 (0.72–1.02)	0.80 (0.67–0.96)	0.93 (0.77–1.13)
Adjusted model[b]	0.92 (0.77–1.10)	0.83 (0.68–1.01)	0.90 (0.73–1.11)

Key message

Offspring exposed to metformin as single adjunctive treatment for gestational diabetes appear to have similar short-term neonatal outcome as offspring exposed to nutrition therapy alone, and more favorable outcome compared with offspring exposed to combined metformin-insulin treatment or insulin alone.

Perinatal Outcomes in Early and Late Gestational Diabetes Mellitus After Treatment from 24-28 weeks' Gestation

Pregnant women with risk factors for diabetes enrolled from before 20 weeks' gestation in the TOBOGM trial

OGTT before 20 weeks' gestation

\+ Early GDM

\- No early GDM

OGTT at 24-28 weeks' gestation

\+ Treated Early GDM (EGDM)

\- No GDM Reference

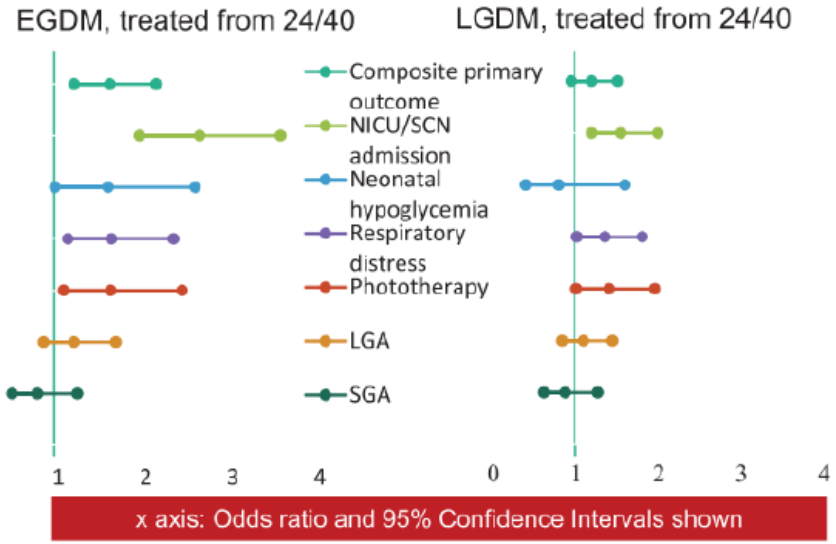
\+ Treated Late GDM (LGDM)

Composite Primary Outcome:
Birth <37^{+0} weeks' gestation; Birth weight ≥4500 g, Birth trauma; Neonatal respiratory distress, Need for phototherapy; Stillbirth/neonatal death, Shoulder dystocia

EGDM (Early GDM): Diagnosed before 20 weeks' gestation but not treated unless persisting at 24-28 weeks' gestation)

LGDM (Late GDM): only present at 24-28 weeks' gestation

Reference: Normal OGTT at 24-28 weeks' gestation



Treatment for early GDM should be initiated before 20 weeks' gestation to avoid the effects of exposure to maternal hyperglycemia from early pregnancy.

20/40, 20 to 40 weeks' gestation; GDM, gestational diabetes mellitus; LGA, large for gestational age; OGTT, oral glucose tolerance test; NICU, neonatal intensive care unit; SCN, special care nursery; SGA, small for gestational age; TOBOGM, Treatment Of BOoking Gestational diabetes Mellitus.

ANAMNESI

Madre con diabete insulino-dipendente. Tamponi VR negativi. TORCH negativo

ALLA NASCITA

38+6 da TC elettivo per posizione trasversa. PN 3290. APGAR 8/9

A circa 10 minuti di vita insorgenza di distress respiratorio ingravescente con necessità di assistenza in nCPAP e ossigenoterapia intorno al 35-40%

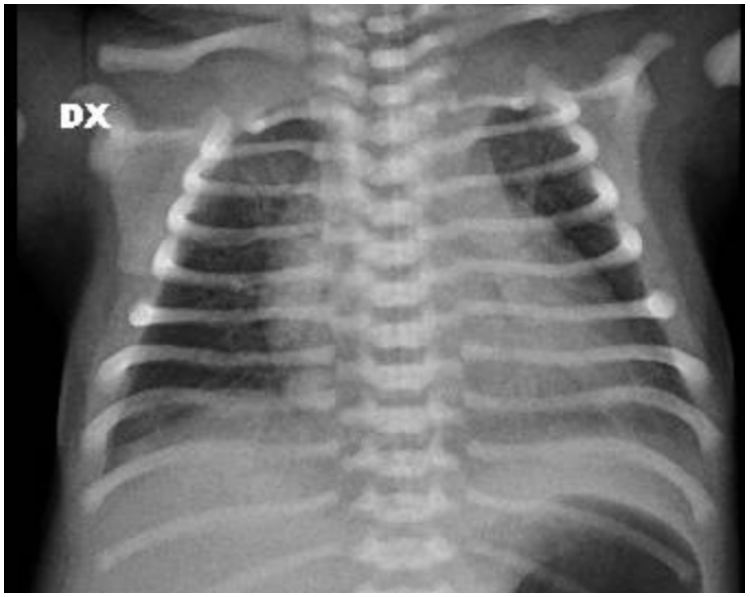
Ipoglicemia

EGA cordonale arterioso non acidosi, Hb 7 gr/dl

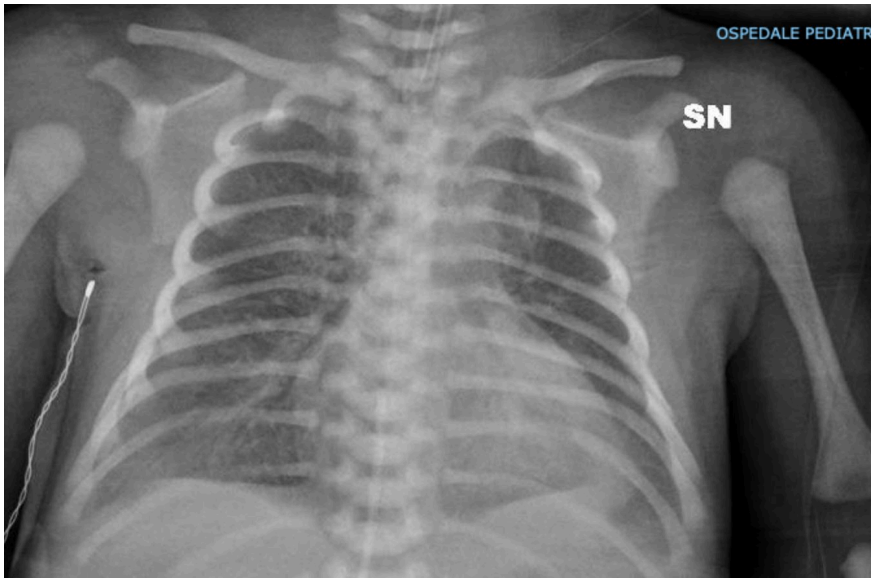
TIN OPBG

TIN OPBG: Quadro respiratorio

All'ingresso



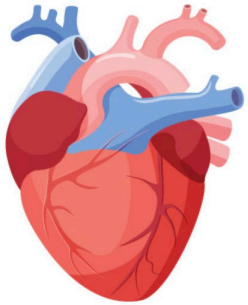
A 12 ore di vita



Insufficienza respiratoria con necessità di intubazione e ventilazione meccanica per 36 ore, poi ventilazione non invasiva (nIPPV,nCPAP, HFNC)

TIN OPBG: Quadro cardiovascolare

All'ingresso



A 36 ore di vita

ECOCARDIOGRAMMA
«…Ventricolo dx dilatato con cinesi conservata
Buona funzione sistolica ventricolare sx
Lieve IT, PSVDx sovrasistemica
Dotto arterioso morfologicamente ampio ma funzionalmente restrittivo per restringimento sul versante polmonare con shunt bidirezionale
→Severa ipertensione polmonare..»

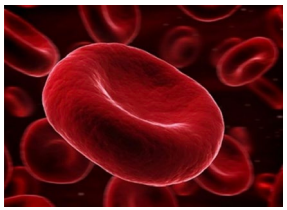
ECOCARDIOGRAMMA
«..V dx dilatato con cinesi ai limiti inferiori/lievemente depressa
Buona la cinesi del Vsx PDA piccolo con shunt bidirezionale a basso gradiente
Conclusioni→PSVDx circa sistemica PDA in chiusura

Severa ipertensione polmonare alla nascita
con successiva comparsa di **disfunzione ventricolare dx** per cui ha iniziato milrinone a circa 36 ore di vita con controlli eco seriati

TIN OPBG: Quadro ematologico

- **Hb all'ingresso in TIN 5gr/dl. Trasfuse emazie con immediato beneficio**
- **Eseguita ecografia encefalo per escludere sanguinamenti: nella norma**

ESAME RICHIESTO: valutazione dell'emorragia feto-materna



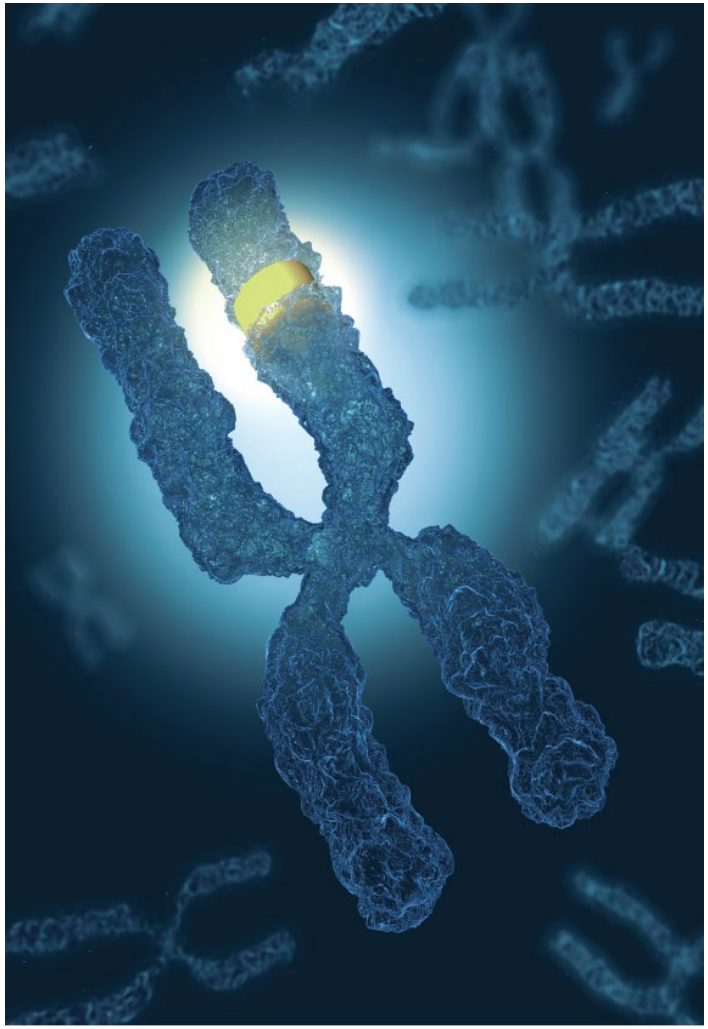
Test in immunofluorescenza:

Emazie HbF^{+} = 4,1 % sul totale delle emazie materne

ml FMH = 90,03 ml (v.n. < 4 mL nel 99% delle gravidanze)

- **A 8 giorni di vita RS in AA**
- **Sospeso milrinone dopo scalaggio in 6 giornata di vita**
- **Alimentazione avviata in 2 giornata di vita e rapido raggiungimento di FEF**
- **Dimesso in 11 giornata di vita, con controllo in follow up a distanza di una settimana (ndr)**


I PRIMI 1000 GIORNI DI VITA:
I GIORNI IMPORTANTI PER LA NOSTRA SALUTE
LE MERAVIGLIE DELL'EPIGENETICA
crea
Ministero della Salute
ccm



UN BUON INIZIO DURA TUTTA LA VITA

Figure 1. A life-course approach for disease prevention and effect on disease prevalence (3).

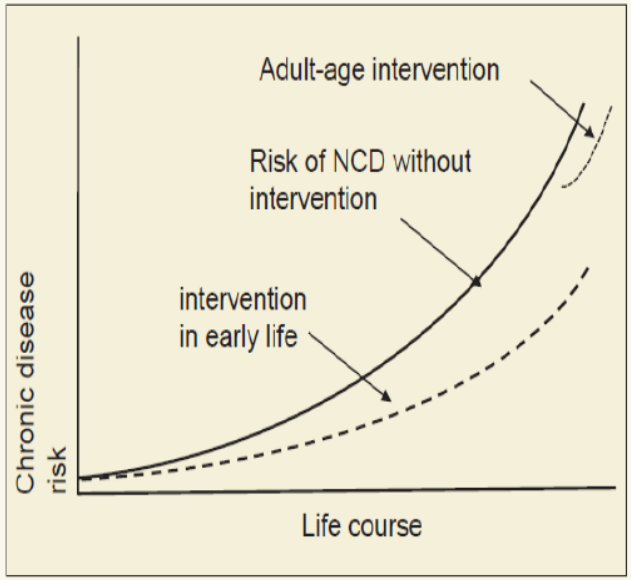
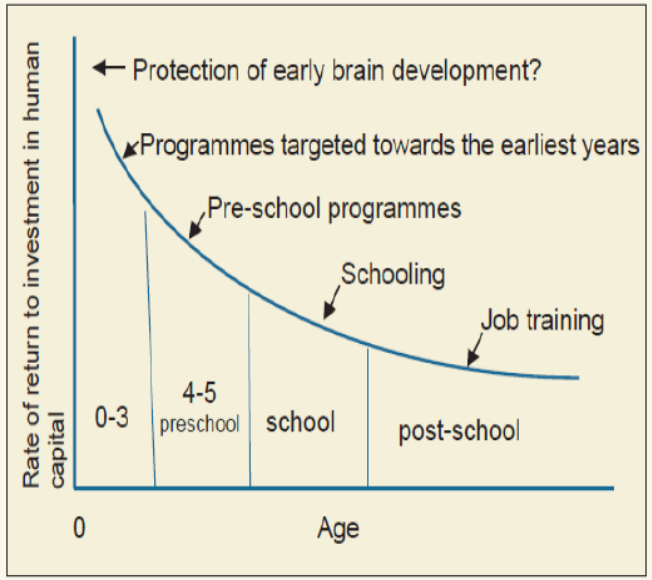


Figure 2. Rate of return of economic and social benefit with interventions at different stages of the life-course (7).



Phillipe Grandjean and Jessica Perry
Entre Nous No.82 – 2015
http://www.euro.who.int/en/healthtopics/Life-stages/sexual-andreproductivehealth/publications/entre-nous/entre-nous.

8 AMAZING BENEFITS OF BREASTFEEDING FOR BABIES

Optimal Nutrition

Human milk contains the right nutrients in the right amounts for baby. The nutrient composition even changes to meet the baby's needs over the course of the feeding, the day, and the infant's lifespan.

Stronger Immune System

Human milk contains maternal antibodies that are passed from mother to baby, substances that weaken or destroy harmful bacteria, compounds that help generate antibodies, and factors that promote the growth of friendly bacteria in the infant's gut.

Lower Risk for Obesity

The risk of overweight in children may be reduced by 4% for every month an infant is breastfed up to 9 months of age. This benefit may continue through the infant's teen and adult years.

Helps Brain Development

Studies suggest that breastfeeding is linked with improved cognitive development. The effects appear to be strongest with exclusive and prolonged breastfeeding.

Reduced Risk for Allergies

During the early days of breastfeeding, human milk contains colostrum, which helps protect the infant's gut from potential allergens and foreign bodies that contribute to allergies. Plus, exclusive breastfeeding for the first six months reduces exposure to potential food allergens.

Decreased Risk of SIDS

Several studies suggest that exclusive or partial breastfeeding reduces the risk for Sudden Infant Death Syndrome (SIDS). Infants who are breastfed may have up to 60% less chance of dying from SIDS than infants who are not breastfed.

Lower Risk for Diabetes

In comparison to formula-fed infants, breastfed infants tend to have a lower risk for developing both type 1 and type 2 diabetes later in life, especially those infants at higher risk of developing the diseases.

Protects Against Heart Disease

Several studies suggest a protective effect of breastfeeding on certain cardiovascular risk factors, such as atherosclerosis, blood pressure, and cholesterol profile, along with other cardiovascular risk factors.

References
American Journal of Clinical Nutrition - http://ajcn.nutrition.org/content/84/5/1043.abstract
Centers for Disease Control and Prevention - http://www.cdc.gov/nccdphp/dnpa/nutrition/pdf/breastfeeding_r2p.pdf
Diabetes Care - http://care.diabetesjournals.org/content/35/11/2215.full.pdf
Epidemiology - http://journals.lww.com/epidem/pages/articleviewer.aspx?year=2004&issue=09000&article=00012&type=abstract
HealthyChildren.org - http://www.healthychildren.org/English/ages-stages/baby/breastfeeding/Pages/Breastfeeding-Benefits-Your-Baby's-Immune-System.aspx
Reuters - http://www.reuters.com/article/2013/12/25/us-breast-feeding-idUSBRE9BO08920131225
WebMD - http://www.webmd.com/parenting/baby/news/20110613/breastfeeding-cuts-sids-risk

Created by:
Michelle Loy, MPH, MS, RDN, CSSD
www.gowellnessco.com

Allattare è bello

perché è...

1 Conoscenza Uno dei momenti più emozionanti e belli è certamente quello della prima poppata. **È un modo straordinario per conoscersi e riconoscersi.** Tutti i sensi del neonato sono impegnati: tatto, vista, olfatto, gusto e udito. Ci si conosce, ci si riconosce, si continua a essere una cosa sola.

2 Complicità Non occorrono tante parole, ma basta osservare una madre che allatta e rendersi conto, dal modo in cui guarda il suo piccolo, che **esiste un linguaggio di sguardi e di gesti** che si unisconо in una armonia con pochi altri esempi simili in natura.

3 Amore L'allattamento è un gesto di amore assoluto in cui la madre dona al figlio se stessa, il suo latte, il suo tempo, la sua attenzione e **trasmette non solo nutrimento ma tutta se stessa.**

4 Gioia L'allattamento è un momento di gioia per il bambino e per la mamma. **La gioia del donare, la gioia del condividere,** la gioia di dare gioia rendono l'allattamento un momento di vera felicità. L'impegno, la fatica e il sacrificio, se vissuti con gioiosità, rendono tutto più bello per la madre e per il bambino.

5 Serenità Allattare è tempo senza tempo. Ovunque avvenga, e può avvenire ovunque, è espressione di serenità. **Madre e figlio sono come avvolti da una nuvola** che li protegge e li isola dal contorno.

6 Salute Poche cose donano salute e positività come l'allattamento. Per la madre è scientificamente dimostrato che l'allattamento è la migliore protezione contro il tumore al seno, ma è anche un modo straordinario di favorire il benessere psicofisico. Per il bambino è uno dei migliori investimenti in salute, benessere e serenità che possa ricevere. **I benefici dell'allattamento sono immediati, ma lasciano segni positivi per tutta la vita.**

7 Sicurezza Nessun **alimento è più sicuro del latte della propria mamma.** Il latte materno ha la corretta composizione per far crescere sano e nel giusto modo ogni bambino, nella sicurezza che nulla può essere migliore.

8 Semplicità Allattare è semplice. Seguendo i consigli, seguendo l'istinto e maturando volta per volta la propria esperienza. **L'allattamento è anche il modo più semplice per far crescere un bambino.** Non richiede null'altro che un posto dove mettersi comodi e tranquilli. Semplice per la mamma, semplice per il bambino.

9 Convenienza L'allattamento **è anche la maniera meno costosa per nutrire un bambino.** Ma il vero guadagno è rappresentato dall'investimento in salute: chi è allattato al seno ha più probabilità di star bene, non solo da piccolo, ma per tutta la vita.

10 Praticità La nascita di un bambino comporta molto impegno e le giornate sono risolte più dense e piene. L'accudimento di un neonato richiede dedizione e molte ore di attività, le più svariate. **L'allattamento al seno, possibile ovunque e in qualsiasi momento,** semplifica molto un'attività così importante come l'alimentazione: nulla di più pratico.

Breastfeeding is not the sole responsibility of women, but society's collective responsibility. It should be protected, promoted and supported by:

Governments and health organisations

- An end to the marketing of formula milk via effective legislation, monitoring and implementation of the Code and development of a legal global treaty.
- Increased regulation and transparency around lobbying to decrease the influence of formula milk companies.
- Investments in maternity protection, supporting breastfeeding at the workplace and enforcing legislation prohibiting discrimination against women during maternity.
- Health organisations rejecting funding from the CMF industry.

Healthcare systems and education

- Healthcare systems to prioritise women-centred and culturally appropriate breastfeeding care and support since pregnancy.
- Improving the breastfeeding education, training and skills of healthcare professionals.
- Empowering parents and families to breastfeed their children for as long as they wish.
- Training providers and families on baby behaviours after birth to prevent unnecessary prelacteals and the early introduction of infant formula, as both practices shorten the duration of breastfeeding.

Breastfeeding, Gestational Diabetes Mellitus, Size at Birth and Overweight/Obesity in Early Childhood

Table 3. Associations between the duration of full breastfeeding and overweight/obesity in early childhood, stratified by size at birth.

Duration	N (%)	OR (95% CI)	aOR (95% CI)
		SGA	
<1 month	103 (7.1)	Ref.	Ref.
1–2 months	22 (7.5)	1.07 (0.66, 1.73)	1.41 (0.78, 2.53)
3–5 months	65 (6.4)	0.90 (0.65, 1.24)	1.03 (0.70, 1.51)
≥6 months	25 (7.9)	1.12 (0.71, 1.76)	0.89 (0.46, 1.70)
		AGA	
<1 month	2732 (18.3)	Ref.	Ref.
1–2 months	491 (18.3)	1.00 (0.89, 1.11)	1.02 (0.91, 1.13)
3–5 months	1539 (13.1)	0.67 (0.63, 0.72)	0.73 (0.68, 0.78)
≥6 months	452 (15.1)	0.79 (0.71, 0.88)	0.84 (0.75, 0.93)
		LGA	
<1 month	867 (31.3)	Ref.	Ref.
1–2 months	130 (28.0)	0.86 (0.69, 1.06)	0.86 (0.69, 1.08)
3–5 months	455 (23.5)	0.67 (0.59, 0.77)	0.66 (0.57, 0.76)
≥6 months	123 (25.6)	0.75 (0.60, 0.94)	0.70 (0.56, 0.88)
P for interaction		<0.001	

Abbreviation: aOR: adjusted odds ratio; AGA: appropriate for gestational age; CI: confidence interval; LGA: large for gestational age; OR: odds ratio; SGA: small for gestational age. Data adjusted for maternal age, maternal educational levels, parity, gravidity, gestational diabetes mellitus status, hypertensive disorders of pregnancy, neonatal gender, gestational age at delivery, mode of delivery, early-pregnancy body mass index, weight gain during pregnancy, and related anthropometric measurements at 9 months.

TAKE HOME MESSAGES

- I primi 1000 giorni dal concepimento determinano gran parte della morbilità a breve e lungo termine
- Il programming metabolico è un elemento fondamentale nello sviluppo e la crescita del nascituro
- **Il counselling prenatale** multidisciplinare con al centro la triade madre-padre-figlio/a, **la prevenzione** e i programmi di **screening precoce** sono alla base della medicina moderna basata sulle evidenze, sulla ricerca, sull'etica e centrata sul paziente

andrea.dotta@opbg.net

Nulla sarebbe più faticoso che mangiare e bere se Dio, oltre che una necessità, non ne avesse fatto un piacere.