Fontainebleau Ballroom, Fontainebleau Hotel

1 **THE MFMU NETWORK RANDOMIZED TRIAL OF FETAL PULSE OXIMETRY** S. L. BLOOM[1], [1]for the NICHD MFMU Network, Bethesda, Maryland

OBJECTIVE: To measure whether knowledge of fetal oxygen saturation, as an adjunct to continuous electronic fetal monitoring, (1) is associated with a significant change in the overall rate of cesarean delivery, including those performed specifically for non-reassuring fetal heart rate pattern and dystocia, and (2) is related to infant condition at birth.

STUDY DESIGN: 5341 nulliparous women at term in early labor were randomly assigned to "open" or "masked" fetal pulse oximetry. In the "open" arm, fetal oxygen saturation values were displayed to the clinician. In the "masked" group, the fetal oxygen sensor was inserted and the values recorded by computer, but the data were hidden. Labors complicated by a non-reassuring fetal heart rate pattern prior to randomization were documented for subsequent analysis. The trial was stopped before the final sample size of 10,000 because the overall cesarean rate (primary outcome) in the masked arm was higher than expected, and there was >90% power to detect the prespecified 15% reduction in the open arm.

RESULTS:

Outcome	"Open" arm n = 2,629	"Masked" arm n = 2,712	*P*-value
Cesarean Rate-Overall	26.3%	27.5%	0.31
Indication:			
Non-Reassuring FHR	7.1%	7.9%	0.30
Dystocia	18.6%	19.2%	0.59
Forceps/Vacuum	14.5%	14.7%	0.76
5-Minute Apgar:			
7 or less	2.9%	3.2%	0.55
3 or less	0.2%	0.1%	0.34
Newborn Seizures	0.1%	0.2%	0.51
HIE	0	0.04%	1.0
Intrapartum Stillbirth	0	0	—
Neonatal Death	0	0.04%	1.0

Similarly, the rate of cesarean delivery, including the overall rate as well as the rates for non-reassuring fetal heart rate and dystocia, were not significantly different in the subgroup of women (n = 2169) with a non-reassuring fetal heart rate pattern prior to randomization.

CONCLUSION: Knowledge of fetal oxygen saturation is not associated with a reduction in the rate of cesarean delivery or improvement in newborn condition.

2 **LMWH VS NO TREATMENT IN PATIENTS WITH PREVIOUS PREECLAMPSIA OR FETAL GROWTH RESTRICTION PLUS HETEROZYGOUS FACTOR V LEIDEN OR PROTHROMBIN GENE MUTATIONS: A RANDOMIZED TRIAL** PARRETTI ELENA[1], MELLO GIOR[illegible][1], FATINI CINZIA[2], TONDI FILIPPO[1], RIVIELLO CHIARA[1], BORRI PATRIZIA[1], S[illegible]ARSELLI GIANFRANCO[1], ABBATE ROSANNA[2], BAHA SIBAI[3], [1]University of Flore[illegible]e, Italy, Department of Gynecology, Perinatology and Human Reproduction, F[illegible]rence, Italy, [2]University of Florence, Italy, Department of Medical [illegible]gica[illegible]Crit-ical Care, Section of Clinical Medicine and Cardiology, Flor[illegible]ce, Ita[illegible], [3]Univer-sity of Cincinnati, Obstetrics and Gynecology, Cincinnati, [illegible]hio

OBJECTIVE: To determine the effect of LMWH [illegible]regn[illegible]cy outc[illegible]mes in women with the heterozygous Factor V Leiden or G[illegible]10A p[illegible]bin gene mutations with a previous history of preclampsia or fe[illegible] growth restriction.

STUDY DESIGN: 305 women who were eith[illegible]eterozyg[illegible]actor V Leiden or G20210A prothrombin gene mutations ca[illegible]iers and with a previous history of preeclampsia or FGR were randomi[illegible]d into [illegible]atment with either dalteparin 5,000 IU/day (n = 151) or no trea[illegible]ent wit[illegible]dalteparin (n = 154). All women were enrolled before 10[illegible]ec[illegible]of g[illegible]. None of the women studied had a personal history [illegible] thr[illegible]embolic disease. The primary outcome of the trial was the rate [illegible] pr[illegible]clan[illegible]or FGR. We estimated that the rate of preeclampsia or [illegible]GR wi[illegible]be 4[illegible]% in the untreated group, and treatment with LMWH w[illegible]sult 33[illegible]uction (from 48% to 32%) in the primary outcome. [illegible]th an [illegible]= 0.05 and a power of 80%, a total of 292 subjects, 146 per gro[illegible] will b[illegible]quired to demonstrate such a difference.

RESULTS: Treatment [illegible]h LMW[illegible] was associated wih significant reduction in rates of overa[illegible]eclamp[illegible]arly onset of preeclampsia, overall FGR and early onset F[illegible]R gro[illegible]h restr[illegible]tion (Table below). In addition, LMWH was associated w[illegible]h [illegible]gher gestaional age (p < 0.01), higher birth weight (p = 0.02), a[illegible]gher m[illegible]an placental weight (p < 0.01).

CONCLUSIO[illegible] LMW[illegible] reduces the recurrence of preeclampsia and FGR in women [illegible]ith the heterozygous Factor V Leiden and G20210A prothrombin gene [illegible]e and with a history of preeclampsia or FGR.

	LMWH group n = 151	No heparin group n = 154	OR (95%, C.I.)
Total preeclampsia	10 (6.6)	44 (28.6)	0.32 (0.18-0.58)
<30 weeks	2 (1.3)	27 (77.5)	0.12 (0.03-0.48)
FGR (no preeclampsia)	14 (9.9)	67 (43.5)	0.28 (0.17-0.46)
<30 weeks	2 (1.3)	48 (31.2)	0.06 (0.01-0.26)

3 **THE ASSOCIATION BETWEEN INHERITED CYTOKINE POLYMORPHISMS AND CEREBRAL PALSY** CATHERINE GIBSON[1], ALASTAIR MACLENNAN[1], PAUL GOLDWATER[2], ERIC HAAN[3], KEVIN PRIEST[4], GUSTAAF DEKKER[5], [1]Adelaide University, Obstetrics and Gynaecology, Adelaide, South Australia, Australia, [2]Women's and Children's Hospital, Microbiology and Infectious Diseases, Adelaide, South Australia, Australia, [3]Women's and Children's Hospital, Department of Genetic Medicine, Adelaide, South Australia, Australia, [4]Department of Health, Epidemiology Branch, Adelaide, South Australia, Australia, [5]Adelaide University, Maternal Medicine, Adelaide, South Australia, Australia

OBJECTIVE: To investigate associations between inherited cytokine polymorphisms and cerebral palsy in a large population-based case-control study.

STUDY DESIGN: Genomic DNA from the newborn screening cards of 443 Caucasian CP cases and 883 Caucasian controls was tested for five cytokine polymorphisms: Tumour Necrosis Factor alpha –308 (TNF-alpha-308), Mannose Binding Lectin –221 (MBL –221), and three polymorphisms in Exon 1 of the Mannose Binding Lectin gene at codons 52, 54 and 57.

RESULTS: At all gestational ages MBL codon 52 (homozygous or heterozygous) was associated with an increased risk of developing quadriplegia (OR 2.74, 0.95-6.96), and MBL codon 54 (homozygous or heterozygous) increased the risk of developing diplegia (OR 1.55, 1.03-2.32). For babies born at term, the risk of developing quadriplegia was associated with heterozygous TNF-alpha (OR 1.82, 1.04-3.15), and MBL codon 52 (homozygous or heterozygous OR 3.24, 0.91-9.42). MBL codon 54 (homozygous or heterozygous) was associated with diplegia (OR 2.12, 1.10-4.05). The presence of any polymorphism in MBL exon 1 at term approximately doubled the risk of developing diplegia (OR 1.94, 1.05-3.62). Carriage of an abnormal TNF-alpha allele was associated with hemiplegia for babies born <32 weeks gestation (OR 2.38, 1.02-5.58). Overall, the presence of any cytokine polymorphism was associated with CP (OR 1.37, 95% CI 1.02-1.84).

CONCLUSION: The data derived from this large population sample of Caucasian cerebral palsy cases and controls demonstrate that carriage of polymorphisms in the TNF-alpha and MBL genes are associated with an increased risk of cerebral palsy.

4 **WHO RANDOMIZED TRIAL OF CALCIUM SUPPLEMENTATION AMONG LOW CALCIUM INTAKE PREGNANT WOMEN** JOSE' VILLAR[1], HANY ABDEL ALEEM[2], MARIO MERIALDI[1], MATTHEWS MATHAI[3], MOHAMED ALI[1], NELLY ZAVALETA[4], MANORAMA PURWAR[5], JUSTUS HOFMEYR[6], NHU NGOC NGUYEN[7], LIANA CAMPODONICO[8], SIHEM LANDOULSI[1], GUILLERMO CARROLI[8], MARSHALL LINDHEIMER[9], [1]World Health Organization, Geneva, Switzerland, [2]Assiut University, Assiut, Egypt, [3]Christian Medical College, Vellore, Tamil Nadu, India, [4]Instituto de Investigación Nutricional, Lima, Peru, [5]Government Medical College and Hospital, Nagpur, Maharashtra, India, [6]University of Witwatersrand, East London, Eastern Cape, South Africa, [7]Hung Vuong Hospital, Ho Chi Minh City, Vietnam, [8]Centro Rosarino de Estudios Perinatales, Rosario, Santa Fe, Argentina, [9]University of Chicago, Chicago, Illinois

OBJECTIVE: To determine if calcium supplementation to low calcium intake pregnant women reduces the incidence of preeclampsia.

STUDY DESIGN: First we verified systematic reviews of randomised trials of prenatal calcium supplementation that suggested such treatment prevents preeclampsia, when dietary calcium intake is deficient. Then a randomized, double blinded trial was conducted through the World Health Organization collaborating institutions in Argentina, Egypt, India, Peru, South Africa and Vietnam, in populations with mean dietary calcium intake <600 mg/day. Nulliparous normotensive women received either 1.5g Ca/day or placebo throughout pregnancy starting before week 20. Primary outcomes were preeclampsia and preterm delivery, secondary were eclampsia, gestational hypertension. After the trial a secondary analysis of composite outcomes stressing severity was performed.

RESULTS: 8,325 women were randomised. Both groups had similar gestational age and demographic characteristics at entry. Compliance (both 85%) and follow-up loss rates were similar (calcium 3.4%, placebo 3.7%). Supplementation was associated with a reduction in preeclampsia starting at gestational week 32, most evident by 35 weeks (p = 0.04), but these changes failed to achieve significance in the overall sample. However reduction in severe preeclamptic complications did (RR = 0.76; 95%CI 0.66-89). Eclampsia and severe gestational hypertension were significantly lower as were adverse outcomes measured through a composite index of severe maternal morbidity and mortality (RR = 0.80; 95%CI 0.70-0.91), and neonatal death (RR = 0.70; 95%CI 0.56-0.88). In a stratified analysis, preterm delivery was only reduced in women <20 years of age (RR = 0.82; 95%CI 0.67-1.01).

CONCLUSION: A 1.5g calcium/day supplement started before midpregnancy may prevent preeclampsia, but most importantly reduces its severity and maternal and/or neonatal morbidity and mortality in low calcium intake women. An easily implemented intervention could prevent preeclampsia-associated morbidity and mortality especially in developing countries.