114 IMPACT OF AN OBSTETRICAL CASE MANAGEMENT INITIATIVE ON NICU COSTS IN A MEDICAID-FUNDED POPULATION CHARLES RITTENBERG[1], SCOTT SULLIVAN[1], M. MENARD[2], AMELIA ROWLAND[1], ROGER NEWMAN[1], [1]Medical University of South Carolina, Charleston, South Carolina, [2]University of North Carolina at Chapel Hill, Chapel Hill, North Carolina

OBJECTIVE: To analyze the impact on NICU costs of a regional perinatal case management initiative to reduce preterm birth (PTB) in a Medicaid-funded population.

STUDY DESIGN: A grant from the state of South Carolina funded a public/private partnership to reduce PTB and associated costs in Medicaid recipients in an 8 county perinatal region. The program consisted of provider education on the program, PTB risks, and PTB reduction strategies; multiple methods to enroll women for telephonic risk assessment as early as possible; general and targeted healthy pregnancy information and 24/7 nurse access for all enrollees; and ongoing patient-centered case management for women meeting predefined high risk triggers (age $<$ 18; multiples; PTL; bleeding $>$ 14 weeks; barriers to care; history of PTB, LBW, or perinatal death). Medicaid claims, birth certificate, and program data were used to compare neonatal outcomes and costs for the intervention (2006) and a control (2004) year. Data on costs, reimbursements, and inflation were obtained from the Medicaid office and the regional perinatal tertiary care center.

RESULTS: There were 6,356 Medicaid deliveries in the region in 2006; 2,111 were referred for telephonic risk assessment; 317 women had high risk triggers and consented to case management. Total Medicaid neonatal costs for the region were $21,395,161; NICU care accounted for $16,246,472 (76%) of this. We previously reported the impact of this initiative to reduce both the frequency and duration of NICU care and unchanged NICU utilization in an adjacent and demographically similar region without the program. The resulting 1824 fewer NICU days in 2006 v. 2004 represent a $2,739,878 reduction in NICU expenditures. Accounting for grant, delivery, and prenatal care costs, the total savings for the year is estimated to be $2,483,674, or $391 per Medicaid delivery in our region.

CONCLUSION: A coordinated program of telephonic risk assessment and case management used to decrease or delay early PTB can significantly reduce NICU utilization and decrease the overall cost of perinatal care.

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115 SEVERE MATERNAL MORBIDITY DURING DELIVERY HOSPITALIZATIONS, NEW YORK CITY, 1995-2003. HEATHER LIPKIND[1], ANNA SFAKIANAKI[1], EDMUND FUNAI[1], DAVID SAVITZ[2], [1]Yale University, New Haven, Connecticut, [2]Mount Sinai School of Medicine, Department of Community and Preventive Medicine, New York, New York

OBJECTIVE: This study was designed to 1) identify pregnancy complications and other risk factors that led to severe maternal morbidity during the delivery hospitalization and to 2) estimate the severe maternal morbidity rates in NYC.

STUDY DESIGN: We used 1995–2003 NYC birth certificates linked to hospital discharge data to identify delivery hospitalizations with maternal diagnoses and procedures that indicated a possible life-threatening diagnosis or a life-saving procedure. We used indicators described in previous work by Callaghan et al (AJOG, 2008) that were developed on a weighted probability sample to examine morbidity in the entire New York City population. The severe morbidity proportion was defined as the number of women with one or more indicators per 1000 deliveries. This is the first time the indicators have been tested on a complete population.

RESULTS: From 1995-2003 there were 1025677 singleton births in NYC. The severe morbidity proportion was 12.8 per 1000 deliveries (greater than twice that of the previously reported rate). Most women that were classified as having a severe morbidity had ICD-9 codes for respiratory failure, transfusion, hysterectomy or eclampsia. Compared to white women, black women had significantly higher odds of severe morbidity (OR) 2.4 (95% CI 2.3-2.5). Women with Medicaid had significantly higher odds of severe morbidity compared to women with private insurance (OR) 1.5 (95% CI 1.4-1.52). Women who delivered by cesarean had four-fold the odds of severe morbidity than those who delivered vaginally (OR) 4.6 (95% CI 4.5-4.8).

CONCLUSION: This study shows that morbidity is disproportionate for certain racial and socioeconomic groups. It is important to understand contributors to life-threatening maternal morbidity in order to improve obstetrical care.

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116 EXPOSURE TO PRENATAL INFLAMMATION AND FETAL BRAIN INJURY: DOES ROUTE OF EXPOSURE AND GESTATIONAL AGE MATTER? MICHAL ELOVITZ[1], JINGHUA CHAI[2], JUAN GONZALEZ[2], BURD IRINA[3], ELLA OFORI[3], [1]University of Pennsylvania, OBGYN; Maternal and Child Health Research Program, Philadelphia, Pennsylvania, [2]University of Pennsylvania, Philadelphia, Pennsylvania, [3]University of Pennsylvania, Pennsylvania

OBJECTIVE: Exposure to prenatal inflammation either from a systemic illness or with chorioamnionitis is associated with adverse neurodevelopment in offspring. This study was performed to elucidate whether systemic or localized (in utero) inflammation has differential effects on the fetal brain and whether the gestational age at the time of inflammatory insult modulates the effect

STUDY DESIGN: CD-1 mice on E15 and E18 were randomized to either systemic iinflammation (IP administration of LPS) or to localized intrauterine inflammation (LIUI)). 6-10 dams per treatment group per gestational age were used. 6 hrs after LPS, amniotic fluid (AF) and fetal brains were collected. Cytokine and genes implicated in neurobehavioral disoders were assessed by QPCR. Cytokine expression in AF was assessed by ELISA.

RESULTS: See table. AF IL-6 levels were 5-fold and 25-fold increased from LIUI on E15 and 18. To systemic inflammation, AF IL-6 was 15-fold and 5-fold increased on E15 and E18. Cytokine mRNA expression was most dramatically increased in setting of LIUI. In response to LIUI, 2/8 neurobehavioral genes assessed were differentially regulated on E15 and 1/8 on E18. To systemic inflammation, 3/8 on E15 were differentially regulated and 2/8 on E18. Pattern of gene expression was divergent between the groups.

CONCLUSION: Exposure to prenatal inflammation, whether systemic or intrauterine, has dramatic effects on gene expression in the fetal brain. Altered gene expression in the fetal brain is varied by the mode of the inflammatory challenge and gestational age of exposure. These studies provide a mechanism by which prenatal inflammation promotes adverse neurodevelopmental outcomes in exposed offspring.

Fetal Brain Cytokine Response (fold change)

Mode of Inflammation	Gestational Day of Exposure	IL-1B	TNF-alpha	IL-6	IL-10
Local	E15	52*	3.1*	2.5*	5.7*
Local	E18	12.4*	2.8*	3.2*	1.2
Systemic	E15	2.3	−2.8	1.7	−1.8
Systemic	E18	7.7*	−1.5	1.5	2.2

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117 MAGNESIUM SULFATE (MGSO4) DOSE AND TIMING, AND UMBILICAL CORD MG++ CONCENTRATION: RELATIONSHIP TO CEREBRAL PALSY (CP) DWIGHT ROUSE[1], [1]for the Eunice Kennedy Shriver National Institute of Child Health and Human Development MFMU Network, Bethesda, Maryland

OBJECTIVE: To assess MgSO4 dose and timing (as actually used) and total umbilical cord blood Mg++ concentration in relationship to CP.

STUDY DESIGN: Data are from a randomized clinical trial of MgSO4 to prevent CP among the offspring of women with anticipated preterm delivery from 24-31 weeks' gestation. The planned dosing regimen was a 6-gm loading dose, 2 gm/hr infusion for up to 12 hrs, discontinuation if delivery not imminent, and re-treatment through 33^6 weeks' gestation with the same regimen prior to delivery. We excluded infants with major congenital anomalies and performed logistic regression analyses adjusted for gestational age at birth.

RESULTS: Of 2,056 children assessed for cerebral palsy, 1,311 (64%) underwent umbilical cord total Mg++ measurement. Median cord Mg++ (meq/L) was 2.6 (interquartile range [IQR] 1.7 −3.4) in the active group (n = 622) vs. 1.6 (IQR 1.4 −1.7) in the placebo (n = 689). Cord Mg++ was negatively but not significantly correlated with CP, adjusted OR 0.76, 95% CI 0.54-1.1. Among 986 MgSO4-allocated pregnancies (median dose 32.0 gm, IQR 29.0-45.1), dose and exposure were characterized as 1) any MgSO4 in last 3, 12, or 24 hrs before delivery; 2) total dose; and 3) duration in the same time intervals. In all of these exposure/dose analyses (n =10), receipt, dose, and duration of MgSO4 were not significantly related to CP risk; however, in all but one the association was in the negative direction (i.e., ↑ MgSO4, ↓ CP). The low (3.7 %) rate of CP among the children in the MgSO4-allocated pregnancies, and the 4.0% rate among those with a cord blood Mg++ measurement limited our statistical power.

CONCLUSION: Our analyses do not support any specific modification of the intravenous MgSO4 dosing regimen that reduced the rate of cerebral palsy in our clinical trial.

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