

THE JOURNAL OF MATERNAL-FETAL & NEONATAL MEDICINE

VOLUME 16 • SUPPLEMENT 1 • OCTOBER 2004

Editors-in-Chief

Gian Carlo Di Renzo Dev Maulik Ola Didrik Saugstad

BOOK OF ABSTRACTS
XIX European Congress of
Perinatal Medicine

Athens, Greece
October 13–16, 2004

**Covered in
Index Medicus
and MEDLINE**



Parthenon Publishing

ISSN: 1476-7058

THE JOURNAL OF MATERNAL-FETAL & NEONATAL MEDICINE

Volume 16 Supplement 1

October 2004

ISSN: 1476-7058

The Official Journal of

The European Association of Perinatal Medicine

The Federation of Asia and Oceania Perinatal Societies

The International Society of Perinatal Obstetricians

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FC2.14.6

EXPOSURE TO LOW OUTDOOR AMBIENT TEMPERATURE IN THE MIDTRIMESTER IS ASSOCIATED WITH LOW BIRTH WEIGHT

Elter K., Emine A., Uyar E., Kavak Z.N

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Aim: To investigate the effect of season on birth weight, and also, to determine the meteorological factor and its specific period of exposure, which may contribute to any seasonal variation in birth weight. **Materials and methods:** Retrospective analysis of 3333 singleton live births after 36 completed weeks of pregnancy. Maternal age, parity, route of delivery, sex and individual meteorological variables for the first, second, and third trimesters of each pregnancy were analyzed by using multiple regression analysis with the birth weight as the dependent variable. **Results:** A seasonal pattern was observed with lowest birth weights in women, who had their last menstrual periods in summer and autumn. Upon multiple regression analysis, sex, parity, mode of delivery, and the temperature, which the mother was exposed in the second trimester were the independent determinants of birth weight. **Discussion:** Exposure to low outdoor ambient temperature in the midtrimester may be associated with low birth weight. Although food availability and infections may partly explain the seasonal variation in birth weight in underdeveloped countries, the contributing factors in developed countries remain unclear (Ann Trop Paediatr 1987;7: 66-71; Soc Sci Med. 1987; 24: 733-9). The effect of heat stress on birth weight in nonhuman species has been addressed by several authors, but very few studies have focused on the effects of environmental temperature on birth weight in humans. Recently, Murray et al. have analyzed the contributing factors to this seasonal variation (Obstet Gynecol 2000; 96: 689-95). They also have suggested that exposure to low temperature in the second trimester might directly result in low birth weight (Obstet Gynecol 2000; 96: 689-95). However, further studies should be performed to analyze the environmental factors affecting birth weight as well as the mechanisms of the relationships. This may help to decrease the perinatal morbidity and mortality associated with low birth weight.

FC2.15.1

PRENATAL PHOSPHORUS (iP) DEFICIENCY SYNDROME IN TYPE I SGA-INFANTS.

Vanhaesebrouck Piet, Verburg Nathalie, De Coen Kris, De Praeter Claudine, Goossens Linde, Smets Koenraad, Verhelst & Zecic Helene & Alexandra

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Aims: Metabolic bone disease (MBD) is a late onset iP deficiency syndrome in very low birth weight infants fed unfortified human milk. However scarce reports consider the effects of fetal growth restriction on iP and calcium levels or bone mineral content in the early neonatal period (1-2). The aim of the study was to evaluate the value of biochemical and haematologic data as markers of early onset iP deficiency in SGA-infants. **Material and Methods:** A case-control study on biochemical [serum iP and calciuria] and haematologic data [nucleated red blood cell (NRBC) and platelet count] during the 1st week of life in 50 type I SGA and 50 AGA-infants matched for gestational age (GA). Mean (\pm SD) GA is 33.7 (\pm 3.2). Mean birthweight (BW) is 1289 (\pm 446) and 2255 (\pm 713) g for the SGA and AGA-group, respectively. In addition 13 type II SGA-infants served as a 2nd control group. Calciuria (mg/dL glomerular filtrate (GF) = $[Ca]U/[creatinine]U \times [creatinine]S$ (1). Birthweight ratio (%) = actual BW/BW at p10 of fetal growth chart \times 100. **Results:** Mean (\pm SD) serum iP [3.9 (\pm 1.4) vs 6 (\pm 1.1) mg/dl], calciuria [1.3 (\pm 0.49) vs 0.4 (\pm 0.28) mg/dL GF], platelet count [111.103 (\pm 78) vs 257.103 (\pm 81) /mm³] and NRBC count (day 1) [142 (\pm 232) vs 6.4 (\pm 7.2) /mm³] were significantly different in SGA-infants compared to paired samples in AGA-controls. Moreover serum iP ($r_s = .60$), platelet ($r_s = .40$) and NRBC count ($r_s = -.43$) were significantly correlated with BW ratio in type I SGA-infants, but not in both control groups. There was also a highly significant correlation between serum iP and platelet count ($r_s = .73$) in the SGA-group and not in AGA nor in type II SGA controls. **Discussion:** Simple laboratory data during the 1st week of life clearly discriminate SGA type I from AGA and type II SGA infants. Our findings are consistent with chronic fetal hypoxia and concomitant prenatal iP deficiency in type I SGA-infants. Phosphate intake is of major importance for growth velocity. We suggest that preterm infants with superimposed IUGR are - as a consequence of prenatal iP deficiency - at greater risk for postnatal stunting and MBD than age-matched 'healthy' preterm infants. These data should be considered in interventional studies on mineral enriched feeding and growth hormone regimens for preterm SGA-infants. **References:** 1. Holland P et al. Lancet 1990;338: 697-701 2. Namgung R et al. Clin Chim Acta 2003;333: 1-11