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Association between maternal selenium status in early gestation and risk of preterm birth

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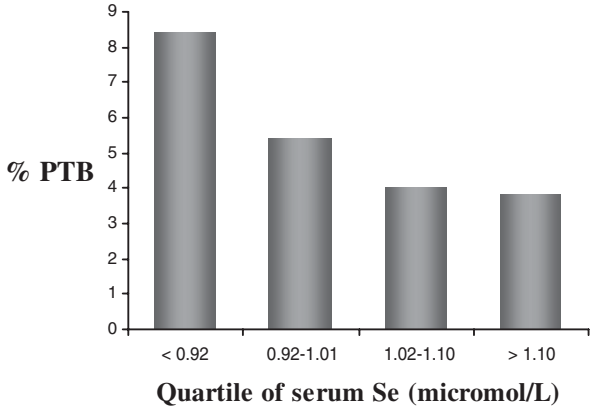
Preterm birth (PTB), defined as birth before 37 weeks gestation, occurs in 5–15% of pregnancies and is the most important cause of perinatal mortality and morbidity(1). Long-term health sequelae include cerebral palsy, respiratory distress syndrome, neurodevelopmental impairment and behavioural problems(2).

Dutch Caucasian pregnant women (n 1197) were followed from 12-weeks gestation in community midwife practices in the vicinity of the city of Eindhoven. Women with thyroid disease, type-1 diabetes or multiple pregnancies were excluded. At delivery, 1129 women had complete data on birth outcomes. Selenium was assessed in 12-week serum samples by dynamic reaction cell ICP-MS. Statistical analysis was performed using the Statistical Package of Social Science (SPSS, 16.0).

Sixty women had PTB: 21 had preterm premature rupture of membranes (PPROM) and 15 had pregnancy-induced hypertension, 13 of whom had pre-eclampsia. Women who delivered preterm had significantly lower serum selenium at 12 weeks gestation than those who delivered at term ($t = 2.9$, $P = 0.001$). The percentages of women with PTB by the quartile of serum selenium at 12 weeks are shown in the figure and are significantly different ($\chi^2 = 8.01$, df = 3, $P<0.05$). Adjusted odds ratios (OR) showed that women in the lowest quartile of serum Se at 12 weeks gestation had twice the risk of having a PTB as the rest (OR 2.01, 95% CI 1.18, 3.14).



To our knowledge, this is the first time that a link has been described between Se status at early gestation and PTB, in particular, to PTB involving PPROM and gestational hypertension or pre-eclampsia. While this may merely be an association rather than a causal effect, we nonetheless acknowledge that selenium is capable of affecting PTB through a number of mechanisms(3): (i) reduced risk of infection and inflammation, (ii) antioxidant and anti-inflammatory effects of selenoenzymes, (iii) vascular effects in the placenta and circulation, (iv) effects on the extracellular matrix and (v) upregulation of haem oxygenase, important for placental endothelial function and uterine quiescence.

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