

CORRESPONDENCE

Atopic manifestations during childhood and fetal exposure to arachidonic acid

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The imprinting of atopic sensitivity remains an intriguing issue, especially with respect to the possible involvement of the early exposures to arachidonic acid (AA) and/or its 'opponents' eicosapentaenoic acid and docosahexaenoic acid. In this Journal, Notenboom et al. [1] recently contributed to this research area by reporting on the relationship between the fatty acid status of women in the third trimester of pregnancy (a proxy for the fatty acid exposure at late fetal development) and atopic manifestations in their children until the age of 6–7 years. The authors did not observe a significant association between early AA exposure and the risk of later atopic manifestations, but recognized that their fatty acid measurements represent fetal fatty acid exposures only in late pregnancy. This is an important issue, as maternal and fetal fatty acid concen-

trations change considerably during pregnancy and fetal development in a fatty acid-specific way [2, 3]. To take these changes into account, more extensive and direct measures of fatty acid exposure over a longer period of fetal development would have been helpful. Such data are available from the Maastricht Essential Fatty Acid Birth cohort and were used by Dirix et al. [4] to investigate associations between the risk of childhood atopy and fetal AA exposure as reflected by the AA concentrations in maternal plasma collected before 16, and at 22 and 32 weeks of pregnancy, and at delivery. In this study, AA levels were also measured in umbilical cord plasma and umbilical arterial and venous vessel walls, representing proxies of fetal AA exposures in late and earlier phases of development, respectively. As shown in Table 1 below, the

Table 1. Odds ratio and 95% confidence interval of associations between arachidonic acid concentration and atopy risk

Fatty acid domain	n [†]	AA [‡] (mean±standard deviation)	OR [§]	95% CI
Maternal plasma 16 weeks GA*	256	9.56±1.46	0.99	0.76–1.30
Maternal plasma 22 weeks GA	237	8.55±1.27	0.79	0.56–1.10
Maternal plasma 32 weeks GA	250	8.11±1.15	0.94	0.66–1.33
Maternal plasma at delivery	252	8.39±1.41	0.83	0.62–1.12
Umbilical plasma	280	16.77±1.62	1.03	0.83–1.27
Umbilical vein wall	185	18.06±1.95	0.96	0.75–1.23
Umbilical artery wall	186	13.8±2.21	0.88	0.68–1.13
Vein-artery differences	183	4.26±2.04	1.10	0.85–1.42

*GA, gestational age.

[†]n, number of observations.

[‡]AA, arachidonic acid. In the multivariable-adjusted regression models, AA was included as a continuous variable (unit: % of phospholipid-associated fatty acids). Thus, the presented odds ratios apply to a 1% increase in the relative AA concentration.

[§]OR, odds ratio. Odds ratios were adjusted for those covariables that, in separate bivariable analyses, were predictors or confounders (i.e. parity, pregnancy duration, season of follow-up measurement, parental atopy history and concentrations of dihomo-gamma-linolenic acid for analyses of AA in maternal plasma; gestational age and concentrations of docosahexaenoic acid for the other analyses).

95% CI: 95% confidence interval.

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relationship between AA levels and atopic manifestations at age 7 was significant for none of the fatty acid domains, as all 95% confidence intervals (CI) included 1. The same was observed for the difference in AA concentrations between umbilical vein and artery vessel walls, a proxy for the fetal AA consumption in late pregnancy.

As the rather narrow CI indicate sufficient power, Dirix et al. [4] concluded that a major impact of early-life exposure to AA on atopic risk at 7 years of age seems rather unlikely. This conclusion is now confirmed by the study of Notenboom and colleagues for atopic risk at 6 years of age.

References

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