Environment International xxx (xxxx) xxx–xxx

Contents lists available at ScienceDirect

Environment International

journal homepage: www.elsevier.com/locate/envint

Dietary acrylamide intake during pregnancy and postnatal growth and obesity: Results from the Norwegian Mother and Child Cohort Study (MoBa)

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ARTICLE INFO

Handling Editor: Olga-Ioanna Kalantzi

Keywords:
Acrylamide
Pregnancy
Postnatal growth
Obesity
MoBa

ABSTRACT

Background: Prenatal acrylamide exposure has been negatively associated with fetal growth but the association with child growth is unknown.
Objectives: We studied the association between prenatal acrylamide exposure and child postnatal growth up to 8 years in the Norwegian Mother and Child Cohort Study (MoBa).
Methods: In 51,952 mother-child pairs from MoBa, acrylamide intake during pregnancy was estimated by combining maternal food intake with food concentrations of acrylamide. Mothers reported their child's weight and length/height up to 11 times between 6 weeks and 8 years. Weight and height growth trajectories were modelled using Jenss-Bayley's growth model. Logistic regression models were used to study the association with overweight/obese status at 3, 5 and 8 years, as identified using the International Obesity Task Force cut-offs. Linear mixed-effect models were used to explore associations with overall growth.
Results: At 3 years, the adjusted odds ratios (95% Confidence Intervals (CI)) of being overweight/obese were 1.10 (1.02, 1.20), 1.12 (1.04, 1.22) and 1.21 (1.11, 1.31) by increasing prenatal acrylamide exposure quartile. Similar dose-response associations were found at 5 and 8 years. Acrylamide intake during pregnancy was associated with higher weight growth velocity in childhood. Children exposed at the highest level had 22 g (95% CI: 8, 37), 57 g (95% CI: 32, 81), and 194 g (95% CI: 110, 278) higher weight at 0.5, 2, and 8 years, respectively, compared to their low exposed peers.
Conclusions: Children prenatally exposed to acrylamide in the highest quartile experienced a moderate increase in weight growth velocity during early childhood that resulted in a moderately increased prevalence of overweight/obesity compared to peers in the lowest quartile. Our study is the first to link prenatal acrylamide exposure and postnatal growth.

1. Introduction

Childhood obesity is a large public health challenge worldwide (de Onis et al., 2010). The major risk factors for obesity are poor nutrition and lack of physical activity. New evidence suggests that exposure to obesogenic chemicals, i.e. chemicals that alter adipogenesis or metabolism, could play a role in obesity development (Heindel et al., 2015; Tang-Peronard et al., 2011). The fetuses and infants may be especially sensitive to exposure to obesogens, even in low concentrations, due to their immature detoxification pathways and developmental plasticity (Janesick and Blumberg, 2012).

Acrylamide is a colourless, odourless, low molecular weight, highly water-soluble organic compound. Acrylamide does not occur naturally and has been industrially produced since the 1950s for various uses, including water and wastewater treatment, as gels in laboratories or in grout for tiling. More recently, it was found that acrylamide can form as a byproduct during the heating of starch-rich foods at high temperatures (> 200 °C), by the Maillard reaction between asparagine and a

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https://doi.org/10.1016/j.envint.2018.01.004
Received 28 August 2017; Received in revised form 10 January 2018; Accepted 10 January 2018

sugar molecule (Dybing and Sanner, 2003; Tareke et al., 2002). In occupationally exposed populations, the main routes of acrylamide exposure are inhalation and dermal absorption, while, in non-occupationally exposed populations, diet is the main source of exposure for non-smokers (Vikstrom et al., 2012). Acrylamide is also found in cigarette smoke, and smoking can contribute extensively to acrylamide exposure (Mojska et al., 2016). According to the Scientific Opinion by the European Food Safety Authority (EFSA), based on data from 24 European countries and approximately 43,000 acrylamide concentrations in foods, the main sources of exposure to adults are fried potatoes, bread, breakfast cereals, biscuits, crackers, crispbread and coffee, and the average exposure was 0.4–1.9 μg/kg body weight/day (EFSA, 2015). After ingestion, acrylamide is extensively absorbed from the gastrointestinal tract, and after reaching the systemic circulation, it is rapidly distributed into the tissues (Zodl et al., 2007). Acrylamide is a known neurotoxicant (Ferguson et al., 2010; IARC, 1994) and can exert reproductive and developmental toxicity effects (Yilmaz et al., 2016). It is classified as "probably carcinogenic" in humans (group 2A) by the International Agency for Research on Cancer (IARC, 1994). In the body, a significant fraction of ingested acrylamide is converted metabolically to the chemically reactive and genotoxic epoxide, glycidamide (Sweeney et al., 2010). Glycidamide is likely to play an important role in the carcinogenicity of acrylamide (Hogervorst et al., 2010).

During pregnancy, 10–50% of dietary acrylamide is transferred via blood through the placenta to the fetus (Annola et al., 2008; Sorgel et al., 2002). Three epidemiological studies have shown a negative association between prenatal acrylamide exposure and birth weight or height or increased risk of having a small for gestational age (SGA) newborn (Duarte-Salles et al., 2013; Kadawathagedara et al., 2016; Pedersen et al., 2012). In Duarte-Salles et al., the adjusted OR for SGA was 1.11 (95% CI: 1.02, 1.21) and the birth weight change was −25.7 g (95% CI: −35.9, −15.4), for the highest vs. the lowest quartile of maternal acrylamide intake (Duarte-Salles et al., 2013). In Pedersen et al., the change on birth weight was −132 g (95% CI: −207, −56), for infants in the highest vs. the lowest quartile of acrylamide hemoglobin adduct (Pedersen et al., 2012). In Kadawathagedara et al., the adjusted OR for SGA was 1.11 (95% CI: 1.03, 1.21) and the change in birth weight was −9.8 g (95% CI: −21.3, 1.7) per 10 μg/day increase in maternal acrylamide intake (Kadawathagedara et al., 2016). Taking into consideration the scarce epidemiological evidence, the CONTAM panel (Contaminant in the Food Chain) of EFSA recommended that further epidemiological studies should be conducted to confirm or refute the inverse relationship between dietary acrylamide intake and impaired fetal growth (EFSA, 2015).

Several epidemiological studies have indicated that small size at birth is a risk factor for a range of metabolic disorders, including higher body mass index (BMI) in adulthood, insulin resistance, increased visceral adiposity and impaired glucose tolerance (Barker, 1998; Calkins and Devaskar, 2011; Gluckman et al., 2008; Stout et al., 2015). However, there is currently no epidemiological study that has examined the potential association between prenatal acrylamide exposure and postnatal growth.

Therefore, the aim of the present study was to investigate the association between maternal dietary acrylamide intake during pregnancy and postnatal growth in children up to age 8 years in a large population-based cohort study in Norway, the Norwegian Mother and Child Cohort Study (MoBa).

2. Material and methods

2.1. Study population

Our study was conducted within MoBa, which is a prospective population-based pregnancy cohort study conducted by the Norwegian Institute of Public Health (Magnus et al., 2016). In brief, participants from all over Norway were recruited by postal invitation prior to their 1st ultrasound visit (17-18th gestational week) during the years 1999 to 2008. The women consented to participation in 40.6% of the pregnancies. MoBa now encompasses 114,500 children, 95,200 mothers and 75,200 fathers. Data used in this study are based on version 9 of the quality-assured data files, released for research in November 2015. All MoBa participants provided written informed consent before enrolment into the study.

The eligible study population included 80,453 women with singleton, live born babies without malformations and chromosomal anomalies and available acrylamide intake estimates. After excluding mother-child pairs with missing information on parity (no missing), maternal age (no missing), maternal education (3% missing), pre-pregnancy BMI (3% missing), gestational weight gain (18% missing), maternal active (1% missing) and passive (1% missing) smoking during pregnancy, maternal alcohol consumption during pregnancy (14% missing), implausible energy intake (i.e. < 4.5 MJ and > 20 MJ, 2% excluded), paternal weight (5% missing), gestational age (0.4% missing), child gender (no missing), birth weight (0.1% missing) and length (3% missing), the population with non-missing information was 52,308 mother-child pairs. Additional mother-child pairs were excluded when no postnatal growth measurement was available, resulting in a final study population of 51,952 mother-child pairs (65% of the source population).

The MoBa study was approved by the Regional Committee for Ethics in Medical Research (S-95113 and S-97045) and the Norwegian Data Inspectorate. The current study was approved by the Regional Committee of Medical Research Ethics for South-Eastern Norway (2016/377).

2.2. Maternal dietary acrylamide intake

The MoBa food frequency questionnaire (FFQ) was used to estimate the daily intake of acrylamide (in μg/day) as previously described in detail by Duarte-Salles et al. (Duarte-Salles et al., 2013) and Brantsaeter et al. (Brantsæter et al., 2008b). In brief, food consumption data assessed by the FFQ, and food contamination data, comprising concentrations of acrylamide in various food items (data from Norwegian, Swedish and European food safety authorities) were combined (Institute for Reference Materials and Measurements, 2005; Livsmedelsverket, 2002; Norwegian Food Safety Authority, 2002; Norwegian Food Safety Authority, 2006; Scientific Committee of the Norwegian Food Control Authority, 2002). Energy-adjusted acrylamide intake (in μg/kcal/day) was calculated by dividing acrylamide intake (in μg/day) by total daily energy intake (kcal). The MoBa FFQ has been validated in 119 pregnant women using a 4-day weighed food record and biological markers as reference methods (Brantsæter et al., 2008a). The validation study demonstrated that it provides valid estimates of dietary intakes and is a valid tool for ranking pregnant women along the distribution of energy, nutrients and foods. The validation study also reported fair agreement between acrylamide metabolite concentrations in 24-hour urine and estimated acrylamide intake (Brantsæter et al., 2008b).

The FFQ contains 225 food items that were aggregated into 100 detailed food groups. Twenty-seven out of 100 food groups contributed to acrylamide intake. In order to identify the main contributors to acrylamide intake during pregnancy, these 27 were further grouped into 12 main food groups. The 12 food groups were: cereals (porridge, cornflakes), bread (white bread, dark bread, rolls), crispbread (crispbread and crackers), pancakes and sweet bakery items (waffle and pancakes, buns, cakes, sweet biscuits), cooked potatoes, fried potatoes, coffee (coffee, decaffeinated coffee, fig coffee, milk based coffee), chocolate, sweets, salty snacks (potato crisps, potato snacks, peanuts and popcorn, pretzels), milk desserts (yoghurt with cereals, chocolate milk and chocolate pudding), and other (poultry, pizza and tacos, breaded fish, olives, dried fruits, chocolate/hazelnut spread).