Colostrum iodine and perchlorate concentrations in Boston-area women: a cross-sectional study

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Summary

Objective To measure levels of colostrum iodine, which has not been previously measured, and perchlorate and cotinine (a surrogate for thiocyanate derived from cigarette smoke) in women up to 60 h postpartum. Perchlorate and thiocyanate are environmental inhibitors of iodide transport into the thyroid and lactating breast.

Design Cross-sectional.

Patients Ninety seven postpartum women in Boston, Massachusetts, USA.

Measurements Colostrum iodine and perchlorate, and spot urine iodine, perchlorate, cotinine and creatinine concentrations were measured.

Results Sufficient colostrum was obtained to measure iodine in 61 samples and perchlorate in 46 samples. Median colostrum iodine content was 51·4 μmol/l (range 21·3–304·2 μmol/l). Perchlorate was detectable in 43 of 46 colostrum samples (median 2·5 μmol/l; range, < 0·05–188·9 μmol/l). Median urine iodine in 97 samples was 82·2 μmol/l (range, 10·3–417·1 μmol/l). Perchlorate was detectable in all 97 urine samples (median 2·6 μmol/l; range, 0·2–160·6 μmol/l). Colostrum iodine content was not significantly correlated with levels of colostrum perchlorate or concentrations per litre of urinary iodine, perchlorate, or cotinine. Colostrum perchlorate concentrations were not significantly associated with urinary iodine, perchlorate, or cotinine levels. Urinary cotinine levels were not significantly associated with urinary iodine or perchlorate levels. There was no association between maternal urinary iodine and urinary perchlorate levels. There was no association between maternal urinary iodine and urinary perchlorate levels. There was no association between maternal urinary iodine and urinary perchlorate levels.

Conclusions Iodine is present in human colostrum and thus available for breastfeeding infants immediately after birth. Perchlorate was also present in 93% of samples measured, but the concentrations did not correlate with colostrum iodine concentrations.

Introduction

Normal thyroid function depends upon sufficient dietary iodine intake. Thyroid hormone plays a vital role in foetal neurodevelopment in utero. Insufficient maternal iodine, particularly in the third trimester and the immediate postpartum period, results in neurological and psychological deficits in children. In infancy, iodine deficiency may result in developmental delays, particularly in language and memory skills. Iodine deficiency is the most frequent cause of preventable mental retardation worldwide.

The breastfed neonate is reliant upon breast milk iodine content. The Institute of Medicine recommends a dietary maternal iodine intake of 174 μmol (220 μg) daily during pregnancy and 229 μmol (290 μg) daily during lactation. Population iodine sufficiency is determined by median urinary iodine concentrations and assumes an average urinary volume of 1·5 l/day and the approximate renal excretion of 90% ingested iodine. Recent national surveys (NHANES I, III, and 2001–02) reported an approximate 50% decrease in median US adult urinary iodine values since the 1970s. Among women of child-bearing age (15–44 year), median urinary iodine decreased from 232·3 to 101·1 μmol/l (294–128 μg/l) between the 1970s and 1990s. The most recent NHANES survey (2001–02) demonstrated that the median urinary iodine concentration in this subgroup remains sufficient and has stabilized at 104·7 μmol/l (132·5 μg/l).

Colostrum, available from the postpartum mammary gland prior to the production of breast milk, is rich in immunoglobulins and nutrients. During lactation, iodine is avidly concentrated in breast milk, due to expression of the sodium/iodide symporter (NIS) in mammary tissue. Man and Benotti in the 1960s measured mean colostrum hormonal iodine by butanol extraction in four postpartum women 3·6 μmol/l (4·5 μg/l). However, we are not aware of any previous studies assessing levels of inorganic iodine, which is the form bioavailable to the nursing infant, in human colostrum immediately after birth. Little is known about iodine nutrition in US breastfed infants and about the factors that influence total iodine content of breast milk. The American Thyroid Association has recently identified as a research priority breast milk iodine concentration measurements in US women and correlation with maternal iodine nutrition and factors such as smoking.

Breast milk iodine content may be influenced by factors other than maternal iodine intake. Perchlorate, a competitive inhibitor of the
NIS present on lactating mammary cells, has been detected in the drinking water of US communities, including Massachusetts, and is found in many foods including cow’s milk. Low-level environmental exposure appears to be ubiquitous in the US population. A recent study measured perchlorate in the breast milk of 36 women from 18 states. Perchlorate was detectable in all samples (range 0.6–92.2 μg/l; 0.6–93.1 μmol/l). Breast milk iodine concentrations were inversely correlated with breast milk perchlorate content in the six samples with perchlorate concentrations > 10 μg/l (> 10 μmol/l). However, we recently reported no correlation between breast milk iodine and breast milk perchlorate content in 57 women, including a subset of 27 women with breast milk perchlorate concentrations > 10 μg/l (> 10 μmol/l).

Cigarette smoke contains cyanide that is metabolized to thiocyanate, a less potent competitive inhibitor of NIS than perchlorate. A recent Danish study demonstrated that cigarette smoking decreases breast milk iodine concentrations proportionate to the amount of maternal tobacco use. Breastfed infants of mothers who smoke have urine cotinine (a nicotine metabolite) concentrations comparable to those in adult smokers. No previous studies have assessed the relationship between either perchlorate or cigarette smoke exposure and the iodine content of human colostrum.

In the present study, we assessed the presence of iodine and perchlorate in human colostrum and sought to determine whether perchlorate and cigarette smoke exposure is associated with decreased colostrum iodine concentrations.

Materials and methods

We obtained colostrum and urine samples from 97 healthy volunteers (mean age 27 ± 6 years; range, 18–42 years) recruited from the inpatient obstetrics unit at Boston Medical Center between October 2006 and April 2007. Subjects were mothers of full-term infants delivered vaginally without the use of iodophor vaginal preparations and who had no known thyroid disease, had never used thyroid hormone replacement or antithyroid medications, and had not received amiodarone within the last 2 years or iodinated contrast dye within the last 6 months. Mothers of infants who were small-for-gestational size (SGA) or large-for-gestational size (LGA) were excluded. The vaginal route of delivery was confirmed, because it is possible that there is a differential use of iodophor skin cleansers (and therefore differing cutaneous absorption of available iodine) in Caesarean section deliveries. The Boston University Medical Center Institutional Review Board approved the protocol and informed consent was obtained from all participants.

We obtained data regarding any history of thyroid disease and participants’ age, race/ethnicity, height, prepregnancy weight, gravity, parity; their infants’ gestational age at delivery, birth weight, and sex; and the date and time of delivery. Self-reported use of or passive exposure to cigarettes was also ascertained (Table 1).

Each subject attempted to provide up to 2 ml colostrum by hand expression within the first 60 postpartum hours (mean 28.6 ± SD 11.2 h; range 9.7–59.3 h) to exclude the collection of transitional milk or mature milk. Sufficient colostrum was available to measure iodine in 61 samples and perchlorate in 46 samples. Spot urine samples were obtained within the same hour as colostrum collection for the measurement of iodine, perchlorate, and cotinine (reflecting cigarette smoke exposure) concentrations.

Total iodine concentrations in colostrum and maternal urine were measured spectrophotometrically. Perchlorate content in colostrum and maternal urine samples was measured using HPLC-mass spectrometry. Maternal urinary cotinine concentrations were measured by immunoassay (Immulite 2000 Nicotine Metabolism Assay; Diagnostic Products Corporation, Los Angeles, CA). Maternal urinary creatinine concentrations were measured by the Jaffe alkaline picrate method (Creatinine_2; Siemens Healthcare Diagnostics, Tarrytown, NY).

Data processing and statistical analyses were performed using Excel 97 (Microsoft, Seattle) and sas version 9.1 (SAS Institute, Cary, NC). Continuous variables were compared across groups using two independent samples t-tests. Ninety-five percent confidence intervals for the difference between means were calculated to assess group differences. All statistical tests were performed at α = 0.05 (2-sided). Pearson’s correlation coefficients and multiple linear regression models were used to assess for relationships between iodine, perchlorate, and other continuous variables.

Results

Median colostrum iodine content in 61 samples was 51.4 μmol/l (range 21.3–304.2 μmol/l). Median urine iodine concentration in 97 samples was 82.2 μmol/l (range, 10.3–417.1 μmol/l). Perchlorate was detectable in 45 of 46 colostrum (median 2.5 μmol/l; range, < 0.05–188.9 μmol/l) and in all 97 urine samples (median 2.6 μmol/l; range, 0.2–160.6 μmol/l). Median maternal urinary cotinine in 95

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Table 1. Subject descriptors (n = 97).

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| Time of colostrum collection from delivery (minutes) | 1716 (mean) ± 673 (SD) |

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available samples was 6·4 nmol/l (range, < 5·7–5481·2 nmol/l), which included six active tobacco users with urinary cotinine levels = 2499·2 nmol/l (consistent with self-report).

Colostrum iodine content was not significantly correlated with levels of colostrum perchlorate ($r^2 = 0·005, P = 0·65$) (Fig. 1), urinary iodine ($r^2 = 0·006, P = 0·54$), urinary perchlorate ($r^2 = 0·004, P = 0·64$) (Fig. 2), or urinary cotinine ($r^2 = 0·009, P = 0·47$) (Fig. 3). Colostrum perchlorate concentrations were not significantly associated with urinary iodine ($r^2 = 0·003, P = 0·71$), perchlorate ($r^2 = 0·001, P = 0·81$), or cotinine ($r^2 = 0·006, P = 0·62$) levels. There was no association between maternal urinary iodine and urinary perchlorate ($r^2 = 0·04, P = 0·06$) or cotinine ($r^2 = 0·002, P = 0·88$) levels. Urinary cotinine levels were not significantly associated with urinary perchlorate ($r^2 = 0·0008, P = 0·79$) levels. There were weak but significant negative correlations between urinary iodine concentrations per gram creatinine and time from delivery ($r^2 = 0·07, P = 0·0081$) (Fig. 4) and colostrum iodine concentrations ($r^2 = 0·12, P = 0·007$).

After exclusion of the six samples collected after 48 h of delivery from the analysis, the results did not change except that there was also a weak significant negative correlation between urinary iodine concentrations (μmol/l) and time from delivery ($r^2 = 0·07, P = 0·01$).

A multiple linear regression model using urinary iodine, colostrum perchlorate, and cotinine levels as cofactors for colostrum iodine concentrations was not significant ($P = 0·79$).

**Discussion**

We are unaware of previous studies assessing human colostrum iodine levels immediately after birth. In one study of lactating mothers in Korea, whose postpartum diet is high in iodine from seaweed, the
mean iodine concentration in human milk at 2–5 days postpartum was 1714 μmol/l (2170 μg/l).\(^{21}\) Mean breast milk iodine 4 weeks postpartum had decreased to 705 μmol/l (892 μg/l). Another recent study of 32 German lactating mothers 5 days postpartum reported a median breast milk iodine concentration of 123 μmol/l (156 μg/l) (range, 26–275 μmol/l, 33–348 μg/l).\(^{22}\) We recently reported a median breast milk iodine content of 123 μmol/l (155 μg/l) (range, 2.1–1555 μmol/l, 2.7–1968 μg/l) in 57 women (median 48, range 10–250 days postpartum).\(^{19}\) Because there is debate regarding the precise timing of the transition from colostrum to breast milk,\(^{22,23}\) the iodine concentrations seen up to 2.5 days postpartum in the present study and those up to 5 days postpartum in the referenced literature are not directly comparable. However, these data do demonstrate that iodine is available to the breastfed neonate immediately after birth.

Our results suggest that exposure to environmental perchlorate, although ubiquitous, does not affect colostrum iodine content. The median urinary perchlorate concentration of 2.6 μmol/l in the present study is comparable to that of the general US population (geometric mean 3.6 μmol/l, 3.6 μg/l).\(^{14}\) Cigarette smoke, generating thiocyanate (a weak NIS inhibitor), similarly does not appear to affect colostrum iodine content, although six only women were cigarette smokers and had truly elevated urine cotinine concentrations. A limitation of the present study is that colostrum iodine concentrations were assessed in a single spot collection. Temporal variability of iodine and iodide-uptake inhibitors such as perchlorate and thiocyanate in breast milk has been described, but the variability of iodine content in colostrum has not been assessed.\(^{22}\) Furthermore, subjects in the present study likely had not ingested anything by mouth during labour and were not consuming their usual diets while hospitalized following delivery (our standard hospital diet does not include iodized salt). Therefore, we cannot make any inferences regarding colostrum iodine content in relation to typical dietary iodine patterns.

There have been only two previous studies which have assessed breast milk iodine and perchlorate levels.\(^{13,15}\) Of those, only our report\(^{15}\) included urinary iodine and perchlorate concentrations, and the median urinary iodine (82.2 μmol/l, range, 10-3–417.1 μmol/l) and urinary perchlorate (2.6 μmol/l, range, 0.2–160.6 μmol/l) measurements in the present study are comparable to those we assessed in women with mature breast milk (90.1 μmol/l, range 19.8–726.8 μmol/l; and 3.0 μmol/l, range 0.37–128.3 μmol/l) (114 μg/l, range 25–920 μg/l; and 3.0 μg/l, range 0.37–127 μg/l, respectively).

Although the American Academy of Pediatrics, United Nations Children's Fund, World Health Organization, and many other health organizations strongly recommend exclusive breastfeeding for infants within the first 6 months of life\(^{26}\) only 11.3% of US women adhere to this guideline.\(^{27}\) We measured iodine and perchlorate content in 17 brands of infant formula.\(^{15}\) Median colostrum iodine concentrations in the present study are slightly lower than those measured in infant formulae (median iodine 114.6 μmol/l, range 66.4–177.0 μmol/l) (145 μg/l, range 84–224 μg/l), although the relative amounts of iodine absorbed by infants cannot be inferred. Our data may serve as a baseline for future studies of trends in human colostrum iodine levels in reference to population iodine nutrition.

Adequate iodine intake for lactating women is important to promote adequate iodine availability to the nursing infant.\(^{28}\) The American Thyroid Association has recently recommended that all women in the US and Canada receive 118.5 μmol (150 μg) iodine supplements daily during pregnancy and lactation and that all prenatal vitamins contain 118.5 μmol (150 μg) iodine.\(^{29}\) We agree and urge that these safe and inexpensive measures be carried out to ensure adequate iodine availability to the breastfed infant.

In summary, this is the first report of the iodine content in human colostrum available immediately to the breastfed infant after delivery, during a period critical for infants' neurodevelopment. Though exposures to environmental perchlorate and cigarette smoke are common, levels of these iodine uptake inhibitors are not associated with decreased colostrum iodine concentrations in this iodine-sufficient study population.

Acknowledgement

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References


