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β-Lactoglobulin secretion in human milk varies widely after cow's milk ingestion in mothers of infants with cow's milk allergy

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Abstract

Background: Cow's milk proteins secreted in human milk may cause cow's milk allergy (CMA) even during exclusive breast-feeding. We studied β-lactoglobulin levels in human milk of mothers of infants with CMA. We also studied intestinal absorption of macromolecules in the same mothers to see whether it is related to the secretion of β-lactoglobulin in human milk.

Methods: CMA was verified with oral challenge in 46 of 55 infants assessed. β-Lactoglobulin levels were assessed in human milk from 53 of 55 mothers of the infants before (basal sample) and 1 and 2 hours after an oral cow's milk load, which was given after a 24-hour milk-free diet. β-Lactoglobulin was determined by an ELISA with a detection limit of 0.002 µg/L. The 6-hour urine recovery of a high-molecular-weight polyethylene glycol (PEG) 3000 was assessed after an oral load of PEG in 45 of 55 mothers.

Results: β-Lactoglobulin was found in the 1- or 2-hour samples in 75% of the mothers. β-Lactoglobulin levels were increased in the 1- or 2-hour samples as compared with the basal levels in about half of the mothers. The respective levels were decreased in one third of the mothers whose basal β-lactoglobulin levels were higher than in the others. β-Lactoglobulin was found in none of the three human milk samples in 15% of the mothers. After an oral load of a high-molecular-weight PEG 3000, the 6-hour urine recovery of PEG was similar in the mothers of the infants with CMA and the mothers of infants without CMA. Neither was the urinary recovery of PEG related to the β-lactoglobulin levels in human milk.

Conclusions: The results support the view that β-lactoglobulin in human milk may contribute to, but does not alone explain, the development of CMA in breast-fed infants. (J ALLERGY CLIN IMMUNOL 1994;93:787-92.)

CMA Cow's milk allergy

PEG Polyethylene glycol

SPT Skin prick test

Key words: [\$\beta\$ -Lactoglobulin](#), [human milk](#), [breast feeding](#), [cow's milk allergy](#), [infants](#), [intestinal absorption](#), [polyethylene glycol](#)

Cow's milk allergy (CMA) in infants may manifest even during exclusive breast-feeding.^{1, 2} This is because human milk of mothers who consume cow's milk contains allergenic cow's milk proteins,^{3, 4} one of the most important of which is β -lactoglobulin.^{5, 6}

In atopic patients increased intestinal permeability to macromolecules has been reported,^{7, 8} but this subject is still controversial.⁹ It is argued that the varying amounts of food antigens in human milk may reflect differences in intestinal absorption of macromolecules.^{10, 11}

We wanted to study secretion of β -lactoglobulin in human milk in mothers of infants with CMA, and therefore we determined β -lactoglobulin levels in human milk before and 1 and 2 hours after an oral cow's milk load in 53 mothers. In addition, to assess whether the secretion of β -lactoglobulin in human milk is related to intestinal absorption of macromolecules in the mother, we measured 6-hour urinary recovery of a high-molecular-weight polyethylene glycol (PEG) 3000 after an oral PEG load in 45 of the mothers.

Methods

Subjects

The subjects consisted of 55 breast-fed infants and their mothers. The infants were examined because of suspected CMA in the hospital ward of the Department of Allergic Diseases of Helsinki University Central Hospital. Their median age was 7.0 months (range, 2.5 to 12.0 months). There were 36 boys and 19 girls. Of the infants, 38 (69%) had a positive family history of atopy; and of the mothers, 25 (45%) had a history of atopy (rhinitis, asthma, atopic eczema). Twenty infants had been exclusively breast-fed, and 35 had also received cow's milk from a median age of 5.0 months (range, 1.0 to 8.0 months). Forty-six infants had received solid foods in addition. The presenting symptom was atopic eczema in 32 (58%), urticaria in 20 (36.5%), and diarrhea in three (5.5%).

The mothers stayed at the hospital with the infants and received hospital food from which the so-called classical allergens (fish, egg, nuts, tomato, and citrus) were eliminated; a 24-hour milk-free diet was introduced before the β -lactoglobulin secretion test.

The mothers gave their informed consent for participation in the study. The study protocol was approved by the Ethical Committee of the Department of Allergic Diseases of the Helsinki University Central Hospital.

Procedures

Skin prick tests (SPTs) (Soluprick, ALK, Horsholm, Denmark) were performed on 55 infants and 43 mothers. Histamine dihydrochloride, 10 mg/ml, was used as positive control, and 50% glycerol was used as a negative control (Soluprick). The reaction was regarded as positive on the appearance of a wheal with a diameter at least 3 mm.¹²

Serum total IgE was determined by Pharmacia IgE radioimmunoassay (Pharmacia, Uppsala, Sweden) in 49 infants.

Serum levels of specific IgE (Phadebas RAST, Pharmacia) for cow's milk and egg were assessed in 55 infants and 41 mothers. RAST values higher than 0.35 Pharmacia RAST units/ml were regarded as positive.

An open cow's milk challenge was performed on 55 infants. After a 3-day milk-free diet, first, a drop of adapted cow's milk formula (Tutteli, Valio, Finland) was placed on the skin (volar arm) and on the lips, and if

there was no reaction, cow's milk was given orally in increasing doses from 5 ml to 100 ml and thereafter ad libitum. The test result was regarded as positive on the appearance of a contact urticarial response to the milk drop, on the appearance of local or systemic symptoms after the oral milk load (usually urticaria or gastrointestinal symptoms), or exacerbation of atopic eczema within 12 hours. The forty-six infants who had positive responses to cow's milk challenge are called hereafter the infants with CMA. Of these infants 16 were solely breast-fed and 30 had also received cow's milk. Results of cow's milk challenge were negative in nine infants.

β -Lactoglobulin secretion in human milk was assessed in 53 mothers. Human milk samples from the mothers were collected before ($n = 47$) (basal sample) and at 1 hour ($n = 52$) (1-hour sample) and 2 hours ($n = 52$) (2-hour sample) after a fat-free cow's milk load of 400 ml, given after a 24-hour milk-free diet. All three samples were obtained from 45 mothers. In some cases an adequate sample collection was not possible. A fraction of each sample was used for human milk challenges, and the rest was stored at -20°C for later determination of β -lactoglobulin levels with an ELISA as described in detail previously.¹³ Briefly, affinity-purified rabbit anti- β -lactoglobulin (0.34 mg/L in 0.05 mol/L carbonate buffer, pH 9.6) was used to coat microtiter plate wells (Immunoplate I, Nunc, Roskilde, Denmark). Plates were incubated for 3 hours at room temperature, followed by an overnight incubation at 4°C . Plates were then washed four times, and free binding sites were blocked with 1.5% gelatin (Bio-Rad Laboratories, Richmond, Calif.). Samples and standards (0.015 to 10 $\mu\text{g/L}$) were tested in a triplicate in several dilutions in phosphate buffer containing 0.5 mol/L NaCl, 0.1% Tween 20 (Bio-Rad), and 3% PEG 6000 (Macrogol; Ph. Nord. NOMEKO SA, Copenhagen, Denmark). The plates were covered and incubated at 4°C overnight and washed four times with washing solution. One hundred microliters of the alkaline phosphatase-conjugated immunoglobulin fraction was used as a second antibody. After an overnight incubation at 4°C and four washes, 100 μL of p-nitrophenol phosphate (1 gm/L in 1 mmol/L diethanolamine buffer, pH 9.8, containing 0.5 mmol/L MgCl_2) was added as substrate. After exactly 30 minutes at room temperature, the reaction was stopped with 50 μL of 3 mol/L NaOH. Absorbances were measured at 405 nm against a blank, and results were calculated from a standard curve. The detection limit of the assay was 0.002 $\mu\text{g/L}$, and the recovery was 93% to 127%. The coefficient of variation (CV) was 5% to 15% within series and 10% between series. The method was specific for β -lactoglobulin when tested with other milk proteins.¹³

Human milk challenge was done on 53 infants. The human milk secreted at 1 hour and at 2 hours after the oral cow's milk load was first placed on the skin and the lips of the infant; then, 2 hours after the oral cow's milk load of the mother, the infant was breast-fed normally (oral challenge). The criteria of positivity were similar to those for cow's milk challenge.

Intestinal absorption of PEG 3000 was studied in 45 mothers. A dose of 5.0 gm of PEG 3000 dissolved in 200 ml of water was given orally, and thereafter urine was collected over the next 6 hours. The urine was measured and stored at -20°C for later determination of PEG with a slightly modified high-performance liquid chromatography.⁸ The dose of PEG recovered in 6-hour urine collection, expressed in percents of the dose administered, was used in the analyses. The high-performance liquid chromatography used (LKB 2152; LKB-Producter, SA, Bromma, Sweden) was equipped with a gel-filtration column (TSK G2000SW, LKB-Producter, SA). The amount of PEG was assayed from the height of the peak against a standard. A control urine collection with added PEG was treated and analyzed in parallel with the samples. Recovery from the urine was around 100%. The within-day coefficient of variation was 0.7% and the coefficient of variation between days was 3% at a concentration of 133 μg and 11% at a concentration of 13 μg standard. Samples produced a coefficient of variation of 20% between days.

Statistics

The distribution of all the data was not Gaussian, and therefore nonparametric statistical analyses were used: the Mann-Whitney U test was used to compare two independent groups, and the Kruskal-Wallis test was used to compare three independent groups.

Results

SPT

SPT reactions to cow's milk were positive in 42 of 48 (91%) infants with CMA and in 1 of 9 (11%) of the other infants. Of the infants with CMA 17 of 45 assessed (38%) had other positive SPT reactions, most often to egg (15 of 43 [35%]). Two of the nine other infants (22%) had positive SPT reaction to egg.

SPT reactions to cow's milk and egg were negative in all of the mothers. In 13 of 43 (30%) of the mothers SPT reactions were positive for at least one of the other allergens (birch, timothy, mugwort, cat, dog); 10 of 13 were mothers of infants with CMA. SPT reactions were positive in 10 of 21 mothers with a positive history of atopy and in 3 of 22 with a negative history.

IgE and RAST

Serum total IgE was increased (>70 kU/L) in 22 of 49 infants assessed (21 of 41 infants with CMA and 1 of 8 others). The median IgE was 72 kU/L (range, 6 to 2500 kU/L) in infants with CMA and 19.5 kU/L (range, 5 to 180 kU/L) in the others.

Cow's milk RAST results were positive for 39 of 45 (87%) of the infants with CMA. One of the nine other infants had a positive cow's milk RAST result. Of the infants with CMA, 34 of 41 (83%) had other positive RAST results, most often to egg (27 of 33 [82%]). Two of nine other infants had other positive RAST results.

RAST results to cow's milk and egg were negative in all of the mothers.

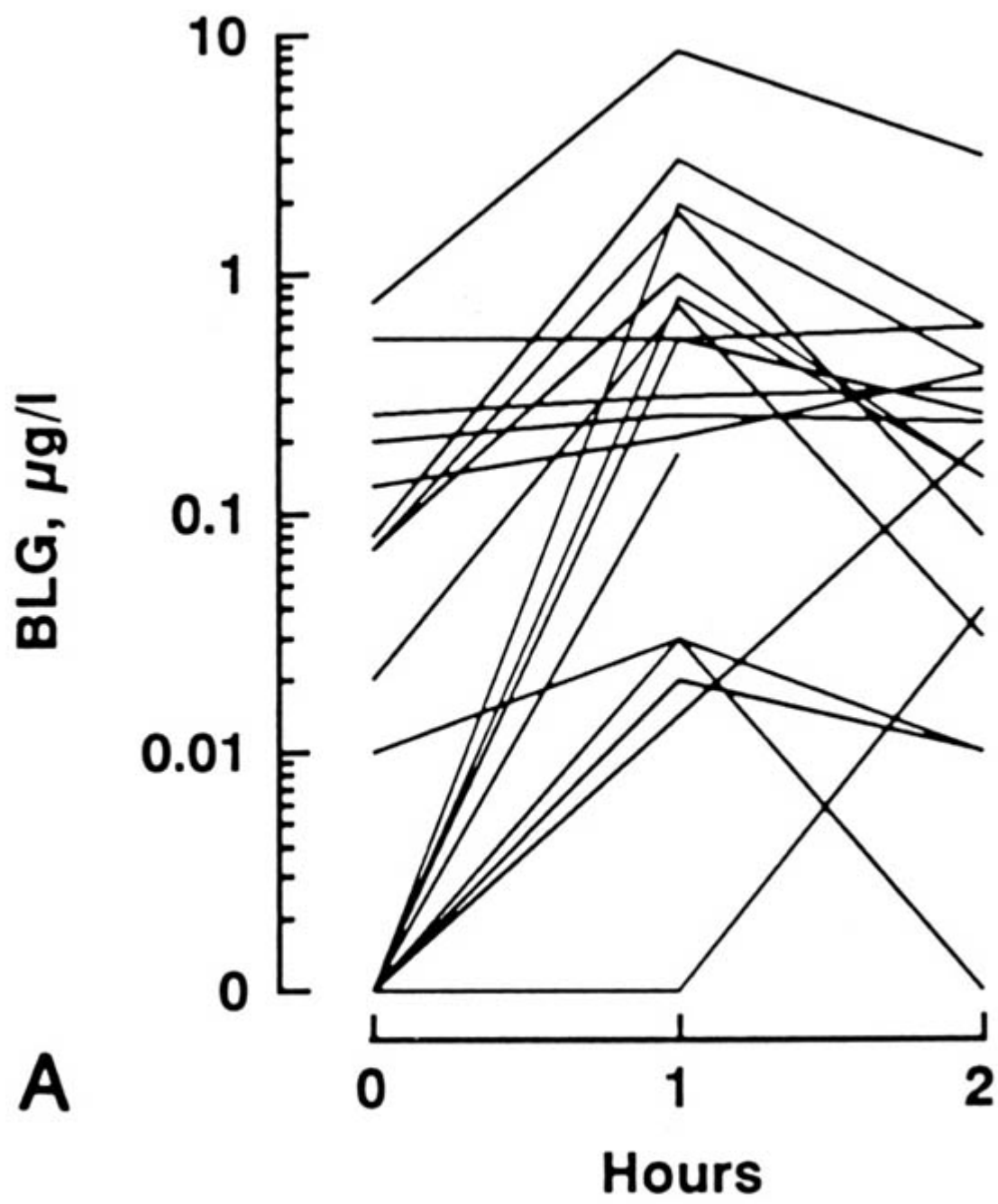
β -Lactoglobulin levels in basal samples

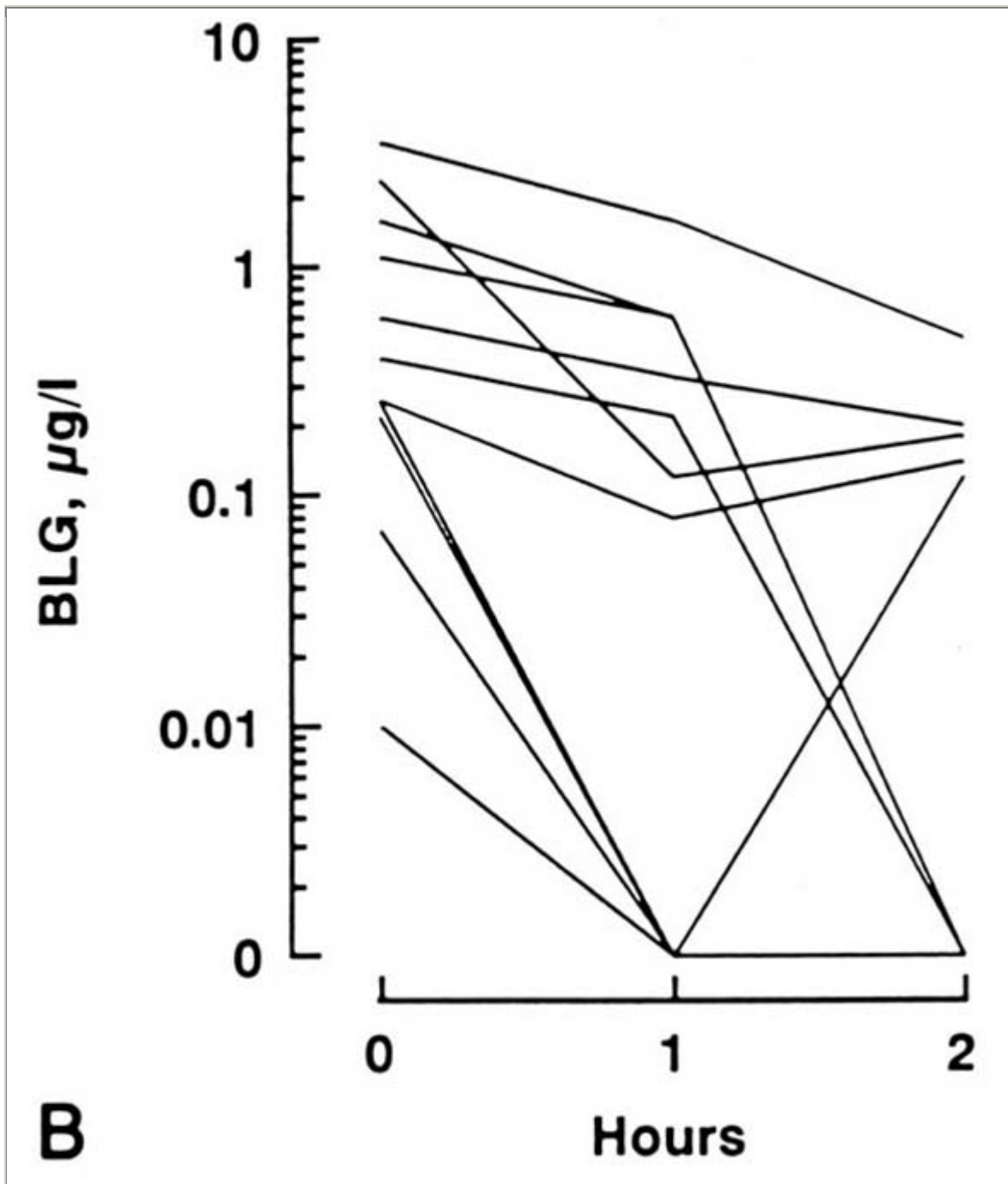
β -Lactoglobulin was found in 23 of 47 (49%) of the basal human milk samples assessed (after a 24-hour milk-free diet, before a cow's milk load to the mother). The median basal β -lactoglobulin level was 0.01 $\mu\text{g/L}$ (range, 0.0 to 3.5 $\mu\text{g/L}$). The basal level was high (>0.5 $\mu\text{g/L}$) in seven samples. All of the basal samples containing detectable amounts of β -lactoglobulin were from the mothers of infants with CMA (23 of 39, 59%). No β -lactoglobulin was found in the eight basal samples from the mothers with infants without CMA.

β -Lactoglobulin levels after an oral cow's milk load

β -Lactoglobulin levels increased in the human milk of 24 of 47 mothers, in 20 of 39 (51%) mothers of the infants with CMA and in 4 of 8 (50%) of the other mothers. The median 0- to 1-hour increase was 0.12 $\mu\text{g/L}$ (range, 0.01 to 7.84 $\mu\text{g/L}$) and 0- to 2-hour increase 0.07 $\mu\text{g/L}$ (range, 0.01 to 2.34 $\mu\text{g/L}$). The increase was high (>0.5 $\mu\text{g/L}$) in eight mothers of infants with CMA and mild in 12 (■ Fig. 1, A). The increase was mild in all four mothers with infants without CMA; the maximal increases (at 1 or 2 hours) were 0.01, 0.01, 0.12, and 0.32 $\mu\text{g/L}$, respectively.

FIG. 1. β -Lactoglobulin (BLG) levels in human milk from mothers of infants with CMA, before and at 1 and 2 hours after ingestion of cow's milk by the mothers. **A**, Data on mothers whose breast milk had an increase in β -lactoglobulin levels ($n = 20$); **B**, data on mothers whose breast milk had a decrease in β -lactoglobulin levels ($n = 13$).





β -Lactoglobulin levels decreased in 13 of 47 mothers, all of whom had infants with CMA (■Fig. 1, B). The basal β -lactoglobulin levels of these mothers were higher ($p = 0.000$); the median was $0.26 \mu\text{g/L}$ (range, 0.01 to $3.50 \mu\text{g/L}$) in these mothers versus $0.01 \mu\text{g/L}$ (0.00 to $0.76 \mu\text{g/L}$) in the mothers with an increase of β -lactoglobulin after the cow's milk load or those with no detectable β -lactoglobulin in any samples.

β -Lactoglobulin levels remained unchanged in the human milk samples from 10 of 47 mothers, and none of their three milk samples had detectable levels of β -lactoglobulin. Six of 39 were mothers of the infants with CMA, and in 4 of 8 were mothers of infants without CMA.

β -Lactoglobulin levels and maternal history of atopy

The median basal β -lactoglobulin level was similar in atopic ($n = 22$) and nonatopic mothers ($n = 25$), $0.05 \mu\text{g/L}$ (range, 0.00 to $2.40 \mu\text{g/L}$) versus $0.00 \mu\text{g/L}$ (range, 0.00 to $3.50 \mu\text{g/L}$). The 1-hour levels tended to be higher in the atopic mothers ($p = 0.093$), $0.22 \mu\text{g/L}$ (range, 0.00 to $8.60 \mu\text{g/L}$) ($n = 23$) versus $0.01 \mu\text{g/L}$

(range, 0.00 to 3.00 µg/L) ($n = 29$), respectively. The 2-hour levels were similar, 0.00 µg/L (range, 0.00 to 3.10 µg/L) ($n = 24$) versus 0.01 µg/L (range, 0.00 to 6.80 µg/L) ($n = 28$), respectively.

Human milk challenges

Ten of forty-six infants with CMA also had a positive reaction to human milk challenge. Three infants had urticaria on skin contact with the human milk secreted 2 hours after the mothers' oral cow's milk load. The β -lactoglobulin levels of these human milk samples were 0.00 µg/L, 0.03 µg/L, and 0.14 µg/L. Only one of the three and seven other infants (with negative skin test reactions to human milk) had a positive response to oral human milk challenge. The maximal β -lactoglobulin levels (basal, 1-hour, or 2-hour) in the human milk from the mothers of these eight infants with a positive oral human milk challenge response were 0.00, 0.02, 0.03, 0.03, 0.26, 0.66, 0.80, and 3.00 µg/L, respectively.

β -Lactoglobulin levels were high (>0.5 µg/L) in some of the samples (basal, 1-hour, or 2-hour) in 15 of 53 mothers (range, 0.54 to 8.6 µg/L). All were mothers of infants with CMA. Only two of the infants had a positive response to oral human milk challenge.

PEG

The median urinary PEG level was 6.5 mg (range, 0.0 to 24.5 mg) (Table I). The urine of one mother did not contain PEG at all. The median recovery was 0.13% (range, 0.0% to 0.49%). PEG recovery was similar in the mothers of infants with CMA and in the other mothers. It was also similar in the mothers with a positive or negative history of atopy, as well as in the mothers with positive or negative SPT results ($p = 0.085$).

TABLE I. Median PEG 3000 recovery with ranges in 6-hour urine after ingestion of 5 gm PEG 3000

PEG	<i>n</i>	Percent	
		Median	Range
Mothers of infants with CMA	38	0.14	0.00-0.49
Mothers of other infants	7	0.12	0.00-0.24
Atopic mothers	20	0.12	0.00-0.41
Nonatopic mothers	25	0.14	0.02-0.49
SPT + mothers	13	0.11	0.00-0.41
SPT - mothers	27	0.16	0.02-0.27

Data are percents of the dose administered (5.0 gm)

There was no significant correlation between the urinary recovery of PEG and the basal, 1-hour, or 2-hour β -lactoglobulin levels in human milk ($n = 37$, $r = -0.05$; $n = 42$, $r = 0.17$; $n = 42$, $r = -0.00$). Nor were the 1-hour and 2-hour changes in β -lactoglobulin related to the recovery of PEG.

Discussion

Cow's milk antigens, such as β -lactoglobulin, are transported in human milk,⁴ as are other food allergens (e.g., ovalbumin,^{3, 14} ovomucoid,³ and gliadin).¹⁵ In the present study one third of the infants with CMA had not received cow's milk; that is, sensitization must have occurred through human milk.

Great intra- and interindividual variation in β -lactoglobulin levels has been observed in human milk.¹⁶ β -Lactoglobulin was found in 40% of 232 human milk samples collected at different periods of lactation from 25 mothers.² However, 6 of 25 mothers had no β -lactoglobulin in any sample (detection limit 5 $\mu\text{g/L}$).² In the present study, with a highly sensitive method¹³ with a detection limit of 0.002 $\mu\text{g/L}$, β -lactoglobulin was found in half of the 151 samples. No β -lactoglobulin was found in 13 of 53 mothers (25%).

No relationship has been found between daily cow's milk intake by mothers and β -lactoglobulin levels in human milk samples taken at different periods of lactation.¹¹ In the present study, β -lactoglobulin was found 1 and/or 2 hours after an oral cow's milk load in half of the 104 samples. In a single mother on whom a lengthy test was performed, β -lactoglobulin level was maximal after 1 hour.¹³ In studies in which β -lactoglobulin levels in human milk were measured 4 to 24 hours after a cow's milk load, the maximal β -lactoglobulin level was found after 8 to 12 hours.^{11, 16} Thus even when assessed after an oral cow's milk load, the appearance of β -lactoglobulin in human milk seems to be variable.

The persistence of β -lactoglobulin in human milk also seems to be variable. In some mothers β -lactoglobulin has been found for 3 days² and even for 9 days¹⁰ after elimination of dietary milk. In the present study, after the mothers spent 24 hours on a milk-free diet, half of the human milk samples (basal samples) contained β -lactoglobulin. The level was even high ($>0.5 \mu\text{g/L}$) in 15% (7 of 47). The secretion of β -lactoglobulin in human milk at 1 and 2 hours after oral cow's milk load seemed to have different patterns in the mothers of infants with CMA. About half of the mothers had an increase in the β -lactoglobulin levels. The increase was mild in about 60% of them and high ($>0.5 \mu\text{g/L}$) in 40%. One third of the mothers of infants with CMA had a decrease in β -lactoglobulin levels. The basal milk samples of these mothers had higher β -lactoglobulin levels than basal milk samples of the other mothers. This group of mothers might represent slower metabolizers of β -lactoglobulin: the decreasing levels of β -lactoglobulin observed still reflect the effects of the 24-hour milk-free diet and not yet the effects of the cow's milk load. The third group of mothers, about 15%, had no change and no detectable β -lactoglobulin in any of the three samples. Of the six infants with CMA whose mothers had no detectable β -lactoglobulin in any of the three samples, two were exclusively breast-fed. Either β -lactoglobulin levels undetectable by the present method or more probably other proteins of cow's milk (e.g., casein, albumin, α -lactalbumin, lactoferrin, and immunoglobulins), which were not assessed in the present study, might have caused the allergic symptoms in the infants of these mothers. Also, if prolonged sampling had been done, β -lactoglobulin might have been found in later samples. In addition, anti- β -lactoglobulin antibodies have been found in smaller quantities in the sera of mothers whose infants had symptoms of allergy during the first months of life.¹⁷ The method used in the present study detects β -lactoglobulin with at least two free antibody binding sites. The influence of possible anti- β -lactoglobulin antibodies on the secretion and assessment of β -lactoglobulin in human milk remains to be investigated.

A control group of healthy infants with no symptoms or signs of CMA would have improved the evaluation of the results of the present study. However, it was not possible for us to have healthy infants and their mothers on the hospital ward where we wanted the study to take place (i.e., for a controlled hospital diet). The pattern of secretion of β -lactoglobulin in human milk in mothers of healthy infants remains to be investigated in other studies.

In the young infants in the present study SPT and RAST results for cow's milk were positive in 91% and 87%, respectively, of those with CMA. In studies of older children with CMA, the percentages are lower, 57% and 59%, respectively.¹⁸ In older children CMA may more often involve non-IgE-mediated mechanisms.

Studies on intestinal permeability in patients with atopic eczema continue to be controversial.^{7, 19} Patients with chronic urticaria or angioedema have been reported to have higher intestinal permeability.²⁰ In the present study mothers with positive and negative histories of atopy or positive and negative SPT results did not differ significantly with regard to urinary recovery of PEG. Thus our results do not suggest that intestinal permeability of macromolecules is increased in atopic patients.

No significant differences in urinary recovery of PEG were found between the mothers of the infants with CMA and the mothers of infants without CMA. Neither did we observe any relationship between the recovery of PEG and the β -lactoglobulin levels in human milk. Thus our results do not support a role for increased absorption of macromolecules in mothers who produce human milk with high β -lactoglobulin levels.

In conclusion, we did not find any difference in intestinal absorption of macromolecules (PEG 3000) between the mothers of infants with CMA and the mothers of infants without CMA. Neither was urinary recovery of PEG related to β -lactoglobulin levels in human milk. But we did find β -lactoglobulin in the human milk of 75% of mothers after an oral cow's milk load. The mothers of infants with CMA had different patterns of secretion of β -lactoglobulin in human milk. About half of them had an increase in β -lactoglobulin in 1- or 2-hour samples after ingestion of cow's milk, one third had a decrease, and 15% had no detectable β -lactoglobulin in any of the three samples analyzed. The results support the view that β -lactoglobulin in human milk may contribute to, but does not alone explain, the development of CMA in breast-fed infants.

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A father's IgE-mediated contact urticaria from mother's milk

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February 2004 • Volume 113 • Number 2

Primary prevention of atopic dermatitis in breast-fed infants: What is the evidence?

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Cow's milk challenge through human milk evokes immune responses in infants with cow's milk allergy

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Kirsi-Marjut Järvinen, MD, Soili Mäkinen-Kiljunen, MSc, Hanna Suomalainen, MD

Efficacy and safety of hydrolyzed cow milk and amino acid–derived formulas in infants with cow milk allergy

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Erika Isolauri, MD, Yelda Sütas, MD, Soili Mäkinen-Kiljunen, MSc, Simo S. Oja, MD, Riitta Isosomppi, MSc, Kristiina Turjanmaa, MD