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Food antibodies in milk from Guatemalan women

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HUMAN MILK contains antibodies against a variety of microorganisms and their products[1-3] that seem to protect the breast-fed infant by preventing adhesion of pathogens or their toxins to the mucosa.[3, 4] Antibodies directed against food components have also been found in milk[5]; these antibodies may prevent food allergies by hindering the intestinal absorption of intact immunogenic food proteins by the neonate.[6] Since the specific immune response in the mammary gland seems to reflect the mother's intestinal antigenic experience,[1] we have tested milk samples obtained from Guatemalan women for the presence of antibodies against food products.

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MATERIALS AND METHODS

We studied milk specimens obtained from 30 Guatemalan women of three different socioeconomic groups. Ten of the mothers (rural poor) lived in Santa María Cauqué, a rural, traditional community in the highlands;

Abbreviations used
ELISA: enzyme-linked immunosorbent assay
SIgA: secretory immunoglobulin A

ten (urban poor) lived in habitational settlements, post-1976 earthquake, and ten (urban privileged) belonged to high-income families in Guatemala City. The dietary habits varied among the three groups: the rural and urban poor mothers do not consume cow milk; black beans are in more common use among the urban groups than in the rural one. The poor mothers, both rural and urban, consume soy products regularly.

The mothers were recruited as part of a collaborative World Health Organization study of the nutritional quality of human milk.[7] The volume of milk produced in a 24-hour period was estimated by weighing the children before and after every meal. This method has been proven

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Table. Daily output of SIgA and IgA antibodies (% of reference) by Guatemalan women, one month after delivery

	Rural poor (n = 10)	*Urban poor (n = 10)*	*Urban privileged (n = 10)*
SIgA (gm)	0.30*	0.28	0.40
	(0.13 – 0.53)†	(0.03 – 0.69)	(0.17 – 0.81)
IgA antibodies against			
Cow milk	5	4	15‡
	(3 – 12)	(2 – 7)	(5 – 41)
Black beans	32	39	49§
	(13 – 74)	(17 – 63)	(18 – 108)
Soybeans	60	62	43
	(31 – 92)	(24 – 191)	(16 – 140)

*Geometric mean.
†Range.
‡Higher than the two poor groups, $P < 0.01$.
§Higher than the rural poor group, $P < 0.05$.

to be accurate and sensitive.[7] The samples (20 to 30 ml) for immunologic analysis were obtained one month after delivery by means of a manual breast pump (Lopuco Breast Pump Co.), from one breast, two hours after the test weighing session was over. Milk samples were kept frozen at −20°C in glass containers. Immediately before their analysis, the specimens were freed of fat and cells by one cycle of centrifugation at 800 × *g*.

The enzyme-linked immunosorbent assay, as described by Sohl-Åkerlund et al.[4] was used to quantitate milk secretory immunoglobulin A. Levels of specific IgA antibodies directed against cow milk, black beans, and soybeans were determined by means of a modification of the ELISA.[8] The antigens were whole cow milk, a phosphatase buffer solution–extract of cooked black beans, and a protein isolate from soy beans, respectively. The detection antibody was alkaline phosphatase-labeled anti-human colostral IgA rabbit globulin (DaKopatts). Daily antibody production was estimated on the basis of the 24-hour milk volumes and expressed as percent of a reference serum standard. This serum, from a patient with allergy to multiple food products, contains extremely high levels of IgA anti-cow milk and other antibodies. Statistical analysis was done using the Wilcoxon rank sum test.

RESULTS

Because of the great variability of antibody levels within the groups of mothers, the results are presented as geometric means.

The three groups of mothers produced comparable amounts of SIgA in a 24-hour period ($P > 0.1$, Table). The daily output of milk IgA antibodies against cow milk was significantly higher in the samples from urban privileged mothers ($P < 0.01$) than in those from urban poor and rural women. The urban privileged mothers also had significantly higher antibody levels against black beans than the rural ones ($P < 0.05$). There were no differences ($P > 0.1$) in the levels of anti-soybean antibodies among the three population groups.

DISCUSSION

Our results show that Guatemalan women from urban and rural settings produce milk SIgA in comparable amounts. The levels of specific antibodies vary, presumably because of differences in antigenic exposure at the intestinal level: poor women do not consume cow milk, whereas the urban privileged mothers do; the consumption of black beans is also more common among urban privileged populations, whereas soy products are preferentially eaten by the rural and urban poor mothers. These findings support the notion that antibodies found in breast milk reflect the mother's intestinal antigenic experience.[1] If anti-food protein antibodies contribute in the prevention of allergies, as has been suggested,[9] it may be valid to expect untoward reactions among rural and urban poor children fed cow milk and milk products. These negative effects could be prevented if, before delivery, the mothers were induced to produce milk antibodies by feeding them cow milk products.[10]

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Resolution of the need for continuous nocturnal feeding in a patient with severe type I glycogen storage disease

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TYPE I GLYCOGEN STORAGE DISEASE results from deficient activity of hepatic, renal, and intestinal glucose-6-phosphatase. It is accompanied by glycogen storage in these organs and a variety of other derangements: hepatomegaly, hypertriglyceridemia, lactic acidosis, hyperuricemia, hypophosphatemia, and growth failure.[1] Most of these features are apparently caused by frequent and prolonged periods of hypoglycemia, since treatment aimed at maintenance of normal blood glucose concentrations corrects most other features of the illness. However, in order to achieve this treatment goal, infusion of a glucose-containing solution, either via a nasogastric or gastrostomy tube, is necessary every night.[2]

The purpose of this report is to document that one patient with severe GSD-I during infancy and adolescence responded optimally to nocturnal feedings, with catch-up growth and normalization of blood chemistry values, and after six years of treatment no longer requires nocturnal feedings. In addition, she has improved her response to a glucose load and to prolonged fasting.

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Abbreviation used
GSD-I: type I glycogen storage disease

CASE REPORT

This 23-year-old white woman was diagnosed as having GSD-I at age one year. During the first 16 years of life she was treated according to published recommendations, with frequent meals day and night.[2] However, she required hospitalization several times a year because of acidosis and hypoglycemia, and maintained marked elevation in blood concentrations of uric acid (12 to 19 mg/dl), triglycerides (2,100 to 5,610 mg/dl), and lactate (6.2 to 16.1 mmoles/L). In the Table are listed several chemical findings at age 16, during the first admission to the Clinical Research Center at Vanderbilt Medical Center. Her height was fiftieth percentile for 8 years, weight fiftieth percentile for 10 years, and during the past year she had grown only an average of 0.01 cm per month. Treatment with continuous nocturnal feeding of a liquid formula containing 98% of the nonprotein calories as glucose oligosaccharides was begun (Vivonex, Eaton Labs, Inc., Norwich, N.Y.) This infusion was given over a 12-hour period during the night and consisted of about one-third of the total