

*From the Medical Department of P.T. Stanvac Indonesia,
Jakarta*

Lactose Intolerance in an Indonesian Closed Community

by

PEDRO C. SAN DIEGO (*) and AGUS ISKANDAR (**)

Lactose Intolerance is now a recognized cause of gastrointestinal symptoms seen in the practice of occupational medicine. Lactase deficiency in adults with resulting lactose intolerance is the most common of the disaccharidase deficiency syndromes. Individuals involved frequently have diarrhea, abdominal distention and cramps when they take large quantities of milk, the principal carbohydrate of which is lactose. Lactase deficiency seems to be the rule rather than the exception in the majority of the world's population especially among Orientals. Studies on Japanese, Chinese, Filipinos, Indians, Singaporeans and Thais have shown its high incidence (Bolin et al. 1968; Davis and Bolin, 1967; Chung and McGill, Bolin et al. 1970; McDonagh, 1970).

This study was conducted to find out its incidence among Indonesians in a closed community.

Materials and Methods

Two hundred employees from the Lirik, Central Sumatera camp of P.T. Stanvac Indonesia were selected for this study.

Fortytwo patients were from Java, One hundred-fiftyseven from Sumatera, and one from Madura. There were 179 males and 21 females. The ages ranged from 17 to 54 years with a mean of 35.5 years. None of these patients had previous gastrointestinal complaints.

The lactose tolerance test (LTT) was done in the following manner: Each patient was told to fast from midnight. Fifty grams of lactose in 500 ml of water were given orally the next morning. The blood glucose (capillary method) was determined immediately before the lactose intake and then subsequently after 15, 30, 60, 90 and 120 minutes. During the procedure, they were asked about the appearance of abdominal cramps,

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* Medical Direktor, Jakarta.

** Senior Medical Officer, Lirik (Central Sum.).

bloating and/or diarrhea. An elevation of the blood glucose of 20 mg% or more was considered normal.

Results

The ideal response for lactase deficiency is a combination of blood glucose elevation of less than 20 mg% and gastrointestinal symptoms or diarrhea and/or gas. There were 78 (34%) out of 200 patients who were positive. However, if the criterion is blood glucose level with or without symptoms the incidence was higher, 108 (54%) out of 200.

Other studies, (Welsh et al. 1967; Littman et al. 1968; Gilat et al. 1970) have shown that glucose rise alone has a better correlation with lactase levels than the presence or absence of symptoms.

This is not surprising, because symptoms might fail to develop in part of any group subjects given a moderate dose of any osmotic laxative.

In our study, the blood types were also correlated (Table 1). No statistically significant difference was found.

TABLE 1:

Blood Type (*)	No. of patients	Positive LTT	%
A	58	27	47%
B	68	38	56%
O	58	13	53%
AB	15	6	40%

(*) - 198 were Rh positive

2 were Rh negative (1A and 1B).

It was shown that a positive LTT can be further confirmed by doing a glucose-galactose tolerance test to rule out malabsorption of monosaccharides. This was not done in our study. Newcomer and McGill (1967-1966) showed an enhanced LTT by the use of capillary instead of venous blood. This, together with lowering the lower limit of normal to 20 mg% drastically reduce the incidence of falsely flat tolerance curves and eliminated the need for glucose-

galactose tolerance test. Thus, in general, LTT is a good screening procedure and has been shown to correlate well with lactase enzyme levels as noted in biopsy specimens, especially if used with the capillary method (Welsh, 1966; Bolin et al. 1970; McDonagh, 1970).

Diagnosis

Lactase deficiency or Lactose Intolerance can be demonstrated in five ways:

1. By feeding the patient 1 quart of milk and observe for the symptoms.
2. Do lactose tolerance test by giving 50 gms of Lactose. Draw the blood sample immediately before the procedure, examine the blood sugar at 15, 30, 60, 90, 120 minutes. A rise of less than 20 mgs with symptoms of diarrhea and/or gas indicates Lactase deficiency.
3. By radiological studies after giving patient a Barium-Lactose meal as described by Laws and Neale.

4oz. of micropaquo and 25 gms of lactose given to patient.

1 hour (60 minutes) film of the abdomen taken. Films were classified as abnormal or normal according to

1. dilution of Barium
2. dilatation of small intestine
3. transit time of barium to the colon.

Correlation with lactose tolerance test in adult is good.

4. By peroral biopsy of the Intestinal mucosa of small bowel and assay the actual lactase content.
5. By measuring the specific radioactivity of Carbondioxide in the exhaled breath after oral administration of Lactose I-C after an overnight fast.

The test measures the activity level of the Intestinal Enzyme

lactase (Dr. Sasaki-John Hopkins Medical Institute - Baltimore).

Advantage

1. Easier on patient.
2. Yield more information than intestinal biopsy.

Disadvantage

1. Can not be used in Diabetic patient and with Pulmonary Dysfunction. Fever and exercise influence the result.

Pathophysiology

Normally ingested lactose is splitted into its two monosaccharide portions-glucose and galactose when acted upon by the lactase enzyme before it can be absorbed through the intestinal mucosa.

In lactase deficient individuals, lactose is not digested so it remains in the lumen. This draws osmotically large amount of fluid within the gut causing distention, increased peristalsis and abdominal cramps. Colonic Bacteria acts on the unabsorbed lactose fermenting into lactic acid with production of CO₂, resulting into bloatedness and gaseous distention and frothy diarrhea. There are probably two lactases: the major one found in the intestinal mucosa brush border and the second, a non-specific beta-Galactosidesplitting enzyme, concentrated in the cellular cytoplasm which is soluble. The functioning enzyme is located just outside the plas-

TABLE 2 :Racial incidence of Lactose Intolerance or lactase Deficiency.

RACE	COUNTRY	REFERENCE	NUMBER STUDIED	PERCENTAGE LACTOSE INTOLERANCE	ABNORMAL LACTASE LEVEL
Australian	Australian Children (Eliot & Maxwell)	15	19	79%	
New Guinea	New Guinea (Bolin & Davis)	6	8	100%	
American - White	Baltimore-Maryland (Bayless & Rosenweig)	3	20 (adults)	10%	5 %
	Baltimore-Maryland (Huang & Bayless)	21	20 (children)	10%	
	Oklahoma (Welsh et al)	45	145	19%	19.4%
	Chicago, Illinois (Littnan et al)	27	93		
	Rochester Minnesota (Newcomer & McGill)	33	100		6 %
	Baltimore-Maryland (Cuatrecasas et al)	11	19	16%	
Negroes	Baltimore-Maryland (Bayless & Rosenweig)	3	20 (adults)	90%	70 %
	Baltimore-Maryland (Huang & Bayless)	22	20 (children)	35%	
	Oklahoma (Welsh et al)	45	22	77%	

TABLE 2: *Continued*

Negreos (cont'd)	Chicago, Illinois (Littman et al)	27	33	24	110%	75 %
	Baltimore (Cuatrecasas et al)	11	30	41	73%	
	Uganda (Bontu) (Cook & Kajubi)	9	30	68	88%	100 %
	South Africa (Jershy et al)	23		22	90%	
Chinese	United States (Chung & McGill)	38	100	5		100 %
	United States (Huang & Bayless)	22	83	10	95 - 100%	
	Australia (Bolin & Davis)	16	112	30	90%	
		31	30		100%	
Greek Cypriots	London (Mc Michael et al)	32	30	17	88%	
Indians	Oklahoma (Welsh et al)	45		3	67%	
	Colombia, SA (Alzate et al)	1	18	24	58.3%	
	Eskimos (greenland) (Gudmand Hoyer & Jarnum)	19		32	72%	

TABLE 2 : Continued

Indonesian	South Sumatera Pendopo Hospital - 1971 (San Diego)	37	78 (adults)	51%	N.B.: 25 mgs considered lowest limit of normal response according to procedure Hagedorn & Jensen.
	Gajah Mada University - Jogjakarta (A. Surjono et al)	40	53 (adults)	90.6%	
	University of Indonesia (1970 - 1972) (Sunoto et al)	39	838 Infant & children with chronic Diarrhea	52.8%	
			50 healthy pre-school age	72%	
			22 (PCM)	86.4%	
			32 (Post Bowel Surgery)	21.8%	
Israel Jewish	Israel (Rozen & Shafir)	36	93	61%	
Japanese	Japan (Matsunaga)	28	39	75%	
	United States (Chung & McGill)	8	2	100%	
Koreans	United States (Chung & McGill)	8	4	100%	

TABLE 2 : *Continued*

Philippines	United States (Huang & Bayless)	22	10	95-100%
	Philippines (Santos-Ocampo et al)	38	20	45%
Singapore	Singapore (Bolin, Seah et al)	4	98	94%
Thailand	Thailand (adults) (Flatz et al)	17	75	100%
	Thailand (children) (Flatz et al)	17	37	84%
	Thailand (adults) (Troncale et al)	41	39	100%
	Thailand (adults) (Keusch et al)	26	140	97%
	Thailand (children) (Keusch et al)	25	172	57%

ma membrane of the epithelial cells. Its activity takes place within the gut lumen at the very margin of the luminal contents.

Lactase peak activity is present in jejunum and proximal ileum. Low levels have been found in the duodenum and distal ileum. No activity has been found in the stomach or colon. Symptoms vary from each individual with deficient lactase enzyme depending upon the amount of lactose ingested. Some may develop symptoms of diarrhea, gas or abdominal cramps. Some when they ingest not enough milk may or may not develop any symptoms. The degree of lactase deficiency determines the lactose intolerance in an individual.

Discussion

Lactase deficiency is divided into primary (genetic or familial) and secondary (acquired). Secondary lactase deficiency is frequently found with other gastrointestinal disease where there is evidence of mucosal damage. These include ulcerative colitis, regional enteritis, non-tropical and tropical sprue, cystic fibrosis and bacterial infections of the gastrointestinal tract. We are interested solely on primary deficiency. These patients are usually free of symptoms even after taking moderate amount of milk.

The intestinal enzyme lactase is present on or within the brush border of intestinal epithelial cells (Crane, 1966; Dahlquist, 1967; Johnson, 1967).

The major concentration is located just outside the plasma membrane of the epithelial cell in the jejunum and proximal ileum. Its activity takes place within the gut lumen at the very margin of the luminal contents. In normal individuals the disaccharide lactose is hydrolyzed by lactase into glucose and galactose. These monosaccharides are then absorbed. However, in lactase deficient individuals the lactose which remains unaffected stays in the lumen. Its osmotic effect leads to a shift of fluids into the intestinal tract. The pH of the stool also decreases due to the production of lactic acid and short-chain fatty acids from the fermentation of lactose by the colonic bacteria.

This leads to abdominal distention, cramps, increased peristalsis and catharsis. The carbon dioxide produced by the fermentation also contributes to bloating and frothy diarrhea. The symptoms appear within 1-3 hours of milk (lactose) ingestion. The severity depends upon the amount of lactose ingested and the degree of lactase deficiency.

Gastrointestinal symptoms are the cause of approximately 6% of the acute conditions producing lost time disability in the working population of the United States. In Asia perhaps it is about 20 - 25%.

There are significant racial differences in the incidence of lactose intolerance. In adult whites from Northwest Europe and U.S.A., the

Incidence has varied from 10 - 18% (Bayless et al. 1966; Cuatrecasas et al. 1965). In Negroes, Indians in the U.S.A., Greek Cypriots, Australian aborigines and New Guinea natives, it ranges from 35-100% (Eliot et al. 1967; Alzan et al. 1969; Bayless & Rosenweig, 1967; McMichael et al. 1966; Welsh et al. 1967).

Asians, Japanese, Filipinos, Chinese, Koreans, Thais, Singaporeans and Indians showed an incidence of 75 - 100%. In our study, 54% were afflicted. Of those with abnormal LTT many were not aware of their intolerance either because they are not milk drinkers, or because some of them had milk discomfort, like flatulence, when they took milk during childhood and since then have avoided it.

The cause of deficiency is not known. Some suggest that it may represent a long lasting or permanent residue of an illness or non-specific injury. In the tropics malnutrition and residence therein have been associated with non-specific small intestinal mucosal changes. Many researchers believe the deficiency to be of genetic origin while others support the adaptation theory.

The arguments of those in favor of the genetic origin are: Welsh, 1970; Ferguson and Maxwell, 1967; Cook and Kajubi, 1966; Bayless and Rosenweig, 1967; Dawson, 1968; Rosenweig, 1971).

1. There is a family history in many patients with the disorder.

2. There is marked racial prevalence in many areas of the world, regardless environmental differences.

3. There are tribal differences in the frequency of lactase deficiency in Uganda.

On the other hand, Cuatrecasas et al. (1965) showed a strong correlation between milk consumption and lactose absorption. Bolin and Dacis (1969) found an association between lack of continued milk drinking after weaning and lactose intolerance in a group of Chinese students living in Australia. Bolin et al. (1971) showed increased lactase activity in rats after prolonged feeding.

The treatment is low lactose diet (Table 3);

The patient should be informed about the problem and advised to limit the intake of milk and other lactose containing products to an amount that they become symptom-free. Products in which lactose is already fermented to lactic acid such as yoghurt, cultured buttermilk and acidophilic milk may be taken. Occasionally they may need a supplement of calcium gluconate tablets to avoid calcium deficiency (McCracken, 1970).

Summary

Two hundred Indonesians were subjected to lactose tolerance test. None of them complained of previous gastrointestinal symptoms and milk intolerance. One hundred-eight of the 200 (54%) showed definite lactase defi-

ciency. This is relatively low as compared to other studies done by several researchers on Orientals.

Lactose intolerance is a mild, clinical entity; essentially in acute self limiting problem in which not only the industrial physicians should be aware of because if it remains unrecognized, it can lead to chronic complaints and can contribute to lost time disability among the working population.

Treatment when recognized is simple.

TABLE 2: *Lactase deficient diet*

FOODS TO INCLUDE	FOODS TO OMIT
<p><u>MILK & MILK PRODUCTS</u></p>	
<p>None; nutramigen and soybean milks used as milk substitutes (only good source of calcium).</p>	<p>All milk of any species and all products containing milk, as skim, dried, evaporated, condensed; yoghurt, cheese, ice-cream, sherbet, malted milk.</p>
<p><u>MEAT, FISH & FOWL</u></p>	
<p>Plain beef, chicken, fish, turkey lamb, veal, pork and ham.</p>	<p>Creamed or breaded meat, fish, or fowl, sausage products, such as wieners, liver sausage, cold cuts containing milk.</p>
<p><u>E G G S</u></p>	
<p>A l l</p>	<p>n o n e</p>
<p><u>VEGETABLES</u></p>	
<p>A l l</p>	<p>All canned or frozen vegetables, or corn curls if lactose is added during processing.</p>

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TABLE 3 : *Continued*POTATOES AND SUBSTITUTES

White & sweet potatoes, jams, macaroni, noodles, spaghetti, rice.

BREADS AND CEREALS

Any that do not contain milk or milk products.

F A T S

Margarine & dressings which do not contain milk or milk products; oils; shortenings; bacon, butter.

S O U P S

Clear soups, vegetable soups, consommés.

D E S S E R T S

Water and fruit ices, gelatin, angel food cake, homemade cakes, pies, cookies made from acceptable ingredients.

F R U I T S

All fresh, canned or frozen that are not processed with lactose.

MISCELLANEOUS

Nuts and nut-butters, unsalted popcorn, olives, pure sugar candy, jelly or marmalade, sugar, corn syrup.

Any creamed, breaded or buttered, french or instant potatoes if lactose is added during processing.

Prepared mizes, such as muffins, biscuits, waffles, pancakes, some dry cereals, instant cream or wheat.

Read labels carefully.

dressings containing milk or milk products, cream, cream cheese.

Cream soups, chowders, commercially prepared soups containing lactose.

commercial cakes and cookies & mixes, custard, puddings, ice-cream made with milk, any containing chocolate.

Any canned or frozen processed with lactose.

Gravy, white sauce, chocolate, cocoa toffee, peppermints, butterscotch, caramels, molasses, candies, instant coffee, powdered soft drinks, monosodium glutamate, some spiceblends, chewing gum.

THIS DIET SHOULD BE SUPPLEMENTED WITH CALCIUM

In all instances, labels should be read carefully and any products which contains milk, lactose, casein, whey, dry milk solids, or curds should be omitted.

Manufacturers should be contacted if there is doubt concerning ingredients in any product.

Adapted from J. amer. diet. Ass. Vol. 43 No. 3: Page 220.

Lactose Free Diet

1. Avoid milk and milk products; milk, cheese, butter, ice-cream, etc.

2. Avoid Yoghurt.
3. Certain liquors have a high lactose content and liquors in general should be avoided.
4. Instant coffees may have a high lactose content and should be avoided.
5. Frozen fried potatoes (label on the package should be investigated) may contain lactose.
6. Use of milk powder like Nestle with no lactose particularly in children.

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