From the Department of Child Health, Medical School, GadjahMada University, Jogjakarta.

Lactose Intolerance among healthy Adults

by

ACHMAD SURJONO, TELUK SEBODO, JATI SUNARTO and MOENGINAH P.A.

Introduction

Lactose occurring naturally only in milk, must be hydrolyzed into glucose and galactose by small-intestinal enzyme lactase in order to be utilized. The production and presence of lactase is in the brush-border of the intestinal epithelial cells and its activity takes place within those cells (Johnson, 1967; Levine and Haft, 1970). The lactase activity is ditributed uniformly throughout the small intestines, except in the duodenum and distal ileum where low levels are found (Auricchio, et al., 1965).

In case of small intestinal lactase deficiency the orally taken lactose passes unsplit to the large intestines and is fermented by the colonic bacterial flora into glucose, lactic acid and carbon dioxide. The unabsorbed lactose will lead flux of fluid into the lumen, probably from its osmotic effect (Auricchio, et al., 1963) or by chloride diffusion from the intestinal wall (Kern and Struthers, 1966). Clinically it produces abdominal cramps, distention, flatulence and diarrhea. The fermentation products also contribute to the resultant acid, bloating and frothy diarrhea.

Interest in the incidence of lactose intolerance has been widely reported on racial differences of peoples. High percentage of incidence in non-Caucasian adults has been observed (Bayless and Rosensweig, 1966; Cook and Kajubi, 1966; Troncale et al., 1967; Huang and Bayless, 1968; McDonagh, 1970; Kretchmer et al., 1971; Luyken et al., 1971).

The incidence of lactose intolerance in Indonesian adults has not yet been fully investigated. An estimation by questionnaire on cow's milk (lactose) intolerence among doctors in Jakarta (Sutedjo, 1971) and among doctors and medical students in Jogjakarta (Surjono et al.,

*) presented at the Scientific Session, Medical Shool, Gadjah Mada University, March 1st., 1972.
Received 24th March 1972.

1971) revealed 20,4% and 19,54% respectively.

The purpose of this study is to present the frequency of lactose intolerance by random sampling among Indonesian adults by giving an oral lactose load.

Material and methods

Fifty three healthy volunteers were studied during the period of September until December 1971, consisting of five doctors and forty-eight 6th grade medical students, ranging in age from 25 to 31 years.

Their health histories and dietary habits were taken, especially concerning symptoms after milk consumption. No diabetic history was found. Ten of the volunteers were milk intolerant according to their complaints.

Lactose tolerance test (LTT) was performed by the method of Hagedorn-Jensen. Fifty grams of lactose (E. Merck) dissolved in 400 ml. of water were administered to fastings individuals. Fingertip capillary blood was obtained before ingestion and after 30, 60, 90 and 120 minutes. The blood was collected in a mixture of sodium fluoride and immediately analyzed for glucose concentration.

A rise of 25 mg. per 100 ml. or more over the fasting blood glucose level was considered normal.

The results of lactose tolerance tests have been shown to correlate well with lactase enzyme levels as noted on biopsy speciments (McMichael et al., 1965; Danovitch, 1966; Bayless and Rosensweig, 1967).

Therefore, subnormal response to LTT (rise of less than 25 mg. per 100 ml.) is compatible with lactose intolerance or lactase deficiency (Poley, 1970; McDonagh, 1970).

Glucose-galactose tolerance tests could not be performed due to lack of facilities.

Results

The average maximum rise in blood sugar of 53 cases after oral lactose load is 11,1 mg. per 100 ml., ranging from 0 — 54 mg. per 100 ml. Range of blood glucose levels in maximum rise of cases can be seen in table.

Out of 53 cases, 48 (90,%) are lactose intolerant as shown by their subnormal response to LTT. Varying degrees of abdominal discomfort, flatulence and diarrhea occur in this group. Mean maximum rise in blood glucose of this group is 7,8 mg. per 100 ml., ranging from 0 to 24 mg.

Five cases are lactose tolerant with an average maximum rise in blood glucose of 38 mg. per 100 ml. (ranging from 28 to 54 mg%).

TABLE : *Relation between range of blood glucose levels in maximum rise and number of cases.*

Maximum rise in blood glucose (mgm. per 100 ml.)	number of cases
less than 10	29
10 — 24	19
more than 25	5
total	53

Ten cases which had previous histories of milk intolerance were proved to be indeed lactose intolerant. Of the remaining 43 cases who could tolerate a glass of milk, 38 (88,4%) revealed subnormal response to LTT.

Out of 13 cases belonging to the milk consuming group, only 1 is lactose tolerant, whereas 4 out of 40 cases of a non-milk consuming group show normal response after LTT.

This result indicates that lactose intolerance can occur in people either with milk drinking or non milk drinking habits.

Discussion

The incidence of abnormal lactose tolerance of our adult subjects being 90,6%, is comparable to studies done by others in coloured races. The prevalence noted among Negroes is : 70% of United States Negroes (Bayless and Rosensweig, 1966); 88% of Bantu tribesmen in Uganda (Cook Kajubi, 1966); 99% of Yoruba group in Nigeria (Kretchmer et. al., 1971); 100% of Bushnegroes in Surinam (Luyken et al., 1971).

In Oriental adults living in the United States, Huang and Bayless (1968) found an incidence of 95%.

Similar findings have been reported by Chung and McGill (1968) in Chinese, Korea and Japanese. Troncale et al. (1967) demonstrated that almost 100% of Thai adults have lactase deficiency.

The etiology of high prevalence of lactase intolerance in adults is not clear. Ferguson and Maxwell (1967) concluded that lactase deficiency is probably a hereditary disease, transmitted as an autosomal recessive. Huang and Bayless (1968) proposed a genetic factor whereby adult type of lactase may not occur after weaning. This is supported by evidence that the ability to digest a lactose decreases with increasing age. Study done on Thai subjects under 3 months old indicated 30% of abnormal lactose tolerance. Data collected during a year on a group of children to whom powdered milk was supplied as a supplement, show-

ed that after 18 months of age the percentage of abnormal lactose tolerance increased from 30 to 70 percent (Varavithya, 1971).

Surjono et al. (1972) found 31,4% among healthy Indonesian neonates had subnormal response to lactose tolerance, whereas in pre-school an incidence of 72% has been observed by Suharjono et al. (1971).

Various authors are in favour of assuming a genetic origin of lactase deficiency regardless of environmental situations, so the incidence to certain races or ethnic groups. In case of certain people, however, high intestinal lactase activity persist throughout life, which does not generally occur.

Inflammatory and degenerative processes involving the small intestinal mucosa often cause a secondary lactase deficiency. This is seen e.g. in celiac disease, tropical sprue, ulcerative colitis, regional enteritis, giardiasis (Bayless and Christopher, 1969; Poley, 1970). After treatment of the primary disease, the lactase activity usually returns to normal values.

In protein-calorie malnutrition a high percentage of lactose intolerance is also found (Bowie et al., 1963; Prinsloo et al., 1969; James, 1970; Sunoto et al., 1971).

From the case histories of our study, it is evident that milk-drinking habit does not affect lactase levels. Similar findings have been reported by Varavithya (1971). Maintenance of an adequate nutritional status by continued milk ingestion during childhood might delay, for a few years, the apparent onset of inadequate lactose digestion (Bayless et al., 1971).

The clinical manifestations of lactose intolerance are extremely variable. Many cases produce nausea, fullness, vomiting, abdominal cramps, flatulence or diarrhea after drinking of milk or a lactose solution. In some cases the symptoms are very mild or even absent; in others they are severe and easily induced by only a small amount of milk. The symptoms probably depend on how much lactose is ingested, how fast it enters the intestine, how severe the enzyme deficiency is and other factors not yet evaluated (Welsh et al., 1966; Littman, 1966).

As seen from our subjects, 38 out of 43 persons who could still tolerate a reasonable amount of milk are 'lactase-deficients' shown by subnormal response to lactose tolerance test. However, milk containg a low quantity of lactose must be considered in certain conditions e.g. in infants with chronic diarrhea, post-gastroenteritis, condition of protein-calorie malnutrion, etc. The other 10 'lactase-deficients' avoid to drink milk because of the unpleasant effects. Previously, this condition was diagnosed as allergic, neurotic or irritable colon syndrome.

High prevalence of lactose intolerance was noted among healthy Indonesian adults, comparable with findings on other non-Caucasians. This incidence may be due to genetic or ethnic predisposition although infectious diseases particularly gastroenteritis, protein-calorie malnutrition and non or low milk consumption during childhood can not be excluded as the etiology of adult lactase deficiency in this country.

Cases withcomplaints of abdominal discomfort or diarrhea after ingestion of a certain amount of milk seem to be lactose intolerant indeed.

Summary and conclusions

Lactose tolerance test was performed on 53 healthy Indonesian adults. It revealed that 48 (90,6%) were lactose intolerant as shown by symptoms after ingestion of 50 grams of lactose and by the subnormal response to lactose tolerance test.

Although high prevalence of lactose intolerance is found, various people can tolerate a certain amount of milk without evidence of uncomfortable affects.

Acknowledgements

We are greatly indebted to Dr. Sutrisno Eram and his staff of the Jogjakarta Province Laboratory for their cooperations; to Drs. Suharjono and Sunoto from the Department of Child Health, Medical School, University of Indonesia, Jakarta, for their valuable suggestions regarding this study.

REFERENCES

1. AURICCHIO, S., RUBINO, A., LANDOLT, M., SEMENZA, G., PRODER, R. : Isolated intestinal lactase deficiency in the adult. Lancet ii : 324 (1963).
2. AURICCHIO, S., RUBINO, A., MURSET, G. : Intestinal glycosidase activities in human embryo, fetus and newborn. Pediatrics 35 : 944 (1965).
3. BAYLESS, T.M. and ROSENSWEIG, N.S. : A racial difference in incidence of lactase deficiency. J amer. med. Ass. 197 :968 (1966). Topics in clinical medicine : Incidence and implications of lactase dieficiency in white Negro populations. John Hopkins med J. 121 : 54 (1967).
4. BAYLESS, T.M. and CHRISTOPHER, N.L.: Disaccharidase dificiency. Amer. J. clin. Nutrit. 22 : 181 (1969).
5. BAYLESS, T.M., PAIGE, D.M., FERRY, G.D. : Lactose intolerance and milk drinking habits. Gastroenterology 60 : 605 (1971).
6. BOWIE, M.D., BRINKMAN, G.L., JANSEN, J.D.L. : Diarrhoea in protein-calorie malnutrition. Lancet ii: 550 (1963).
7. CHUNG, M.H. and McGILL, D.B. : Lactase dificiency in Orientals. Gastroenterology 54 : 225 (1968).
8. COOK, G.C. and KAJUBI, S.K. : Tribal incidence of lactase deficiency in Uganda. Lancet i : 725 (1966).
9. DANOVITCH, S.H. : The syndrome of lactase deficiency. Gen. Pract. 33 : 117 (1966).
10. FERGUSON, A. and MAWELL, J.D. : Genetic etiology of lactose intolerance. Lancet i : 188 (1967).

11. HUANG, S.S. and BAYLESS, T.M. : Milk and lactose intolerance in healthy Orientals. Science 160 : 83 (1968).

12. JAMES, W.P.T. : Sugar absortion and intestinal motility in children when malnourished and after tre 39 : 305 (1970).

13. JOHNSON, C.F. : Disaccharidase, localization in Harmster intestine brushborder. Science 155 : 1670 (1967).

14. KERN, F. and STRUTHERS, J.E. : Lactase dificiency adn lactose intolerance in adults. J. amer med. Ass. 195 : 927 (1966).

15. KRETCHMER, N., RANSOME-KUTI, O., HURWITZ, R., DUNGY, C. and A-LAKIJA, W. : Intestinal absorption of lactose in Nigerian ethnic groups. Lancet ii : 392 (1871).

16. LEVINE, R. and HAFT, D.E. : Carbohydate homeostasis. New Engl. J. Med. 283 : 175 (1970).

17. LITTMAN, A. : Isolated lactase deficiency in the adult. J. amer med. Ass. 195 : 954 (1966).

18. LUYKEN, R., LUYKEN-KONING, F. W.M. and IMMIKHUIZEN, T. : Lactose intolerance in Surinam. Trop. geogr. Med. 23 : 54 (1971).

19. McDONAGH, T.J. : Lactose intolerance in non-Caucasians. Med. Bull. 30 : 277 (1970).

20. McMICHAEL, H.B., WEBB, J. and DAWSON, A.M. : Lactase deficiency in adults. Lancet i : 717 (1965).

21. POLEY, J.R. : Causes of chronic diarrhea in infants and children. Pract. Pediat. 48 : 143 (1970).

22. PRINSLOO, J.G., WITTMANN, W., PRETORIUS, P.J. KRUGER, H. and FELLINGHAM, S.A. : Effect of different sugars on diarrhea of acute kwashiorkor. Arch. Dis. Childh. 44 : 593 (1969).

23. SUHARJONO, SUNOTO, ASWITHA BUDIARSO adn SUTEDJO : Lactose malabsotion in "healthy" Indonesian Pre-School age children. Paediat. Indon. 11: 251-254 (1971).

24. SUNOTO, SUHARJONO, CYSCA M. LEMBONG, ASWITHA BUDIARSO and SAMSUDIN: Lactose loading tets on Protein-Calorie Malnutrition. Paediat. Indon. 13: 43 (1973).

25. SURJONO, A., MOENGINAH, P.A., TELUK SUBODO and JATI SUNARTO: Intoleransi terhadap air susu sapi (lactose) pada mahasiswa Kedokteran U.G.M. Berkala I. Kedokt. Gama 1971.

26. SURJONO, A., SURJANTORO, TONNY SADJIMIN, ISMANGOEN : Lactose tolerance test on Indonesian newborn infants. Paediat Indon. 13 : 11-16 (1973).

27. SUTEDJO : Cow's milk (lactose?) intolerance among Indonesian doctors of the Dr. Tjipto Mangunkusumo General Hospital, Jakarta. Paediat. Indon. 11: 43 (1971).

28. TRONCALE, F.J., KEUSCH, G.T., MILLER, R.H. OLSON, R.A. and BUCHANAN, R.D. : Normal absorption in subjects with non-specific jejunal abnormalities. Brit. med. J. ii : 578 (1967).

29. VARAVITHYA, W. : personal communication (1971).

30. WELESH, J.D., SHAW, R.W. and WALKER, A. : Isolated lactose deficiency producing post-gastrectomy milk intolerance.Ann. intern. Med. 64 : 1252 (1966).