

Lactose tolerance and intolerance in Malaysians

Peter Michael Barling

Abstract: This review explores the digestibility of lactose by Malaysians, and the value of milk and other milk-derived products as sources of appropriate nutrition for Malaysians. Increased calcium intake through consumption of milk is an effective mechanism for increasing calcium uptake from the diet and thereby minimising the risk of development of osteoporosis in later life. Detailed information about rates of lactose intolerance, and adaptation to dietary lactose and its consequences for Malaysians, will help in the formulation of dietary advice, and improve commercial food manufacturing practice and Government policy directed to the minimization of rates of osteoporosis, which presents a substantial morbidity risk to elderly female Asians in particular.

IeJSME 2012: 6 (Suppl 1): S12-S23

Keywords: lactose, lactose non-persistence, breath hydrogen test, gastrointestinal symptoms, milk and dairy products

Introduction

Lactose (β -D-galactopyranosyl-(1 \rightarrow 4)-D-glucose) is found exclusively in milk and some milk products, and is the principle carbohydrate of all mammalian milks, of which human milk contains the highest concentration (70 g/L). Cows' milk and products immediately derived from it (such as yoghurt) are major potential sources of lactose in the Malaysian diet. This present review is primarily concerned with "lactose intolerance" in adult Malaysians, which occurs as a result of a loss of the capacity of the enterocytes of the duodenum to hydrolyse lactose to its constituent monosaccharides (glucose and galactose) after weaning, as a result of a programmed absence of the enzyme lactose phlorizin hydrolase (LPH) on the mucosal surface of these enterocytes.¹ (The equivalent bacterial enzyme to LPH is sometimes called "lactase", but is more usually known as " β -galactosidase". These terms are synonymous). As well as being "lactose intolerant", individuals lacking LPH as adults are confusingly described in the scientific

literature in a variety of terminologies, including having "hypolactasia", or being "lactase non-persisters".

Of particular interest is the potential capacity of Malaysians to get nutritional advantage from consumption of milk and milk products, and the potential ability of many lactose intolerant Malaysians to tolerate, or adapt to, modest levels of milk in their diet. This interest is stimulated by recent evidence that lactose intolerance is an important risk factor for developing osteoporosis. This is partly because individuals who are lactose intolerant tend to avoid consuming dairy products.^{2,3} Milk consumption data from Malaysia confirm that the average daily intake of milk in Malaysia is less than a quarter of that in parts of Scandinavia.⁴ Milk and milk products are by far the most important sources of calcium throughout the world, but provide only 26% of the calcium intake from various food sources for postmenopausal Malaysian women.⁵ Moreover, the average daily intake of calcium in adult Malaysians (around 0.4 g) represents only 40% of the daily reference values for adults of 1 g.^{6,7} Hence the average Malaysian diet is substantially calcium deficient. Moreover there is mounting evidence that lactose in foodstuffs promotes calcium absorption in animals^{8,9} and humans^{10,11} by an unknown mechanism, but that undigested lactose may interfere with calcium absorption.¹² Lactose stimulates phosphate absorption in rats¹³ and promotes magnesium and manganese absorption in healthy infants.¹⁰ Thus, knowledge of the extent of lactose intolerance in Malaysia, its pattern of inheritance, and an understanding of the variability in the potential of Malaysians of different ethnicities to gain beneficial nutrition from lactose, will provide useful information for formulation of effective dietary advice and policies to minimize the rate of osteoporosis among Malaysians. According to Lau and Woo¹⁴, osteoporosis poses a particular high morbidity risk to elderly female Asians. Understanding lactose tolerance and intolerance is also of scientific and anthropological value, and may add useful information relevant to effective planning of other future studies (for example, milk dietary intervention trials).

For Correspondence:

Professor Peter Barling, Division of Human Biology, School of Medical Sciences, International Medical University, No. 126, Jalan Jalil Perkasa 19, Bukit Jalil, 57000 Kuala Lumpur, MALAYSIA

Email: peter_barling@imu.edu.my

Assessment of lactose intolerance

Several methods are available to assess lactose tolerance and intolerance in adults. Each one of these alternatives has its advantages and drawbacks.

Direct assessment

Direct assessment of LPH enzyme activity can be performed on tissue obtained from a small intestinal biopsy. This test is the so-called “gold standard” method for determining lactose tolerance or intolerance. However, the process is highly invasive as it requires an upper gastrointestinal endoscopy. Moreover, the LPH activity in a particular biopsy specimen is not necessarily representative of the general activity in that region of the small intestine¹⁵, influencing the reliability of the test.

Lactose hydrogen breath test

A lactose hydrogen breath test is currently the “secondary gold standard” for assessment of lactose intolerance,^{16,17} as it is sensitive, noninvasive, inexpensive and can be performed in subjects of all ages. In this test, participants drink a lactose bolus. In those who are lactose intolerant, the non-digested lactose will be partially fermented in the colon, to produce short-chain fatty acids (lactate, acetate, propionate and butyrate), together with gases (hydrogen, methane and carbon dioxide). Hydrogen gas will be partially excreted in participants’ breath,^{18,19} and this can be analyzed for H₂ at regular intervals thereafter.²⁰ The dose of lactose and the cut-off point used in the hydrogen breath test are important as they influence the criteria used to assess lactose intolerance. 50 g of lactose is often used as the challenge dose, and permits a clear separation of LPH persisters from non-persisters. The cut-off is a rise in breath hydrogen of greater than 20 parts per million above the fasting level. A greater increase than this is indicative of colonic fermentation of non-absorbed carbohydrate.²¹ However, the test will miss certain non-persisters whose colonic gaseous fermentation products do not include appreciable hydrogen, or instead are

entirely or largely comprised of methane.²² Between 2 and 20% of individuals may lack a hydrogen-producing colonic flora, while approximately 40% of individuals contain significant numbers of methane-producing bacteria in the colon.²³⁻²⁶ Moreover some individuals have a slow small intestine transit time, thus delaying the entry of undigested lactose into the colon. These individuals need to be tested for up to five hours and very few participants are willing to undergo the test for this length of time.²² More than 85% of lactose intolerant subjects (based on the hydrogen breath test) also develop gastrointestinal symptoms after consuming 50 g of lactose as a single dose.²⁷ These symptoms can provide valuable additional diagnostic information.

Plasma glucose test

When lactose is digested in the small intestine, the hydrolysis products, galactose and glucose, subsequently enter the liver where the galactose will be primarily converted into glycogen. Glucose will mostly enter the peripheral bloodstream and induce a prompt rise in blood glucose concentration.²⁰ Lactose-intolerant subjects will not show such a rise, although there may be a smaller and later increase in blood glucose originating from gluconeogenesis of lactate and/or propionate generated from colonic fermentation of lactose. A rise in blood glucose of at least 1.5 mmol/L is indicative of lactose tolerance.²⁸ The specificity and sensitivity of this lactose tolerance test ranges from 76 to 96%.²⁹ The magnitude of the increase in blood glucose is subject to several hormonal influences, thus reducing the reliability of the test compared to the breath hydrogen test.³⁰

Plasma galactose test

A plasma galactose test, in which a lactose bolus is administered with a 500 mg/kg dose of ethanol to prevent the conversion of galactose to glycogen in the liver²⁸ is much more reliable than the plasma glucose test, although the necessary invasive sampling (to obtain sufficient blood for the galactose assay) makes

the test more difficult to administer on large numbers of subjects,³⁰ and the ethanol exposure is inappropriate for Muslims, who form a majority of the population of Malaysia.

Lactose intolerance amongst Malaysians

This topic was unexplored until 2006, when Asmawi *et al.* presented data on the prevalence of lactose intolerance among adults of the three major Malaysian ethnic groups living under similar conditions on the island of Penang.³¹ Three hundred Malaysians were recruited in this study, 100 of each ethnicity. 88% of the Malays, 91% of the Chinese and 83% of the Indians were found to be lactose intolerant. The primary criterion of lactose tolerance/intolerance in this study was the measurement of urinary galactose levels, which would theoretically only be evident in lactose tolerant individuals. However, it has long been known that the urinary excretion of galactose in response to an oral bolus of galactose (of the same magnitude as would be expected to be generated by lactose tolerant individuals in the study by Asmawi *et al.*) varies widely between individuals, and there are clear gender differences.³² Hence the results of that study are unreliable as indicators of lactose intolerance. Moreover, Asmawi *et al.* did not explore the extent of lactose intolerance in any minority ethnic or tribal groups.

In the last year, our group (Yasmin Beng Ooi, Min Yen Tan and myself, unpublished observations) has studied lactose intolerance in Malaysians recruited from staff and students at Universiti Malaysia Sabah, including Indians, Chinese and Malays. Our study also investigated lactose intolerance in a small numbers of subjects from two East Borneo ethnic groups. The participants, who were largely self-selected, received a 50 g bolus of lactose after fasting overnight. The criteria used to assign subjects as lactose tolerant or intolerant were based on the subsequent pattern of breath hydrogen excretion, changes in blood glucose levels, and the self-reporting of gastrointestinal symptoms, and are presented in Table 1. The outcome of this study was that only 4 of

146 Malaysians (3%) were classified as lactose tolerant. While this was a much lower incidence than previously reported,³¹ it accords with the results from adjacent countries. For example, a lactose intolerance rate of 97% has been reported for Thais.³³

Table 1: Criteria of lactose tolerance and intolerance used in the recent study carried out by the authors of this present review.

Lactose tolerance
No significant increase in breath hydrogen (< 20 ppm) AND a significant increase in plasma glucose (> 1.5 mmol/L) AND an absence of any gastrointestinal symptoms.
Lactose intolerance
A significant increase in breath hydrogen (> 20 ppm) OR no significant increase in plasma glucose (< 1.5 mmol/L) OR a report of diarrhea or flatulence OR two or more of any of the following: abdominal pain, flatulence, nausea, cramping or borborygmi.

It can be concluded that rates of lactose intolerance amongst Malaysians are extremely high, probably overall in the region of at least 95%.

The development of lactose intolerance

In exclusively breast-fed babies, lactose is a most important source of energy during the first six months of life. It is completely hydrolyzed to glucose and galactose by LPH. A neonate's LPH is tethered by a trans-membrane domain to the intrinsic microvillus membrane of the external (mucosal) surface of enterocytes at the tips of the intestinal villi in the jejunum, which is the first segment of the small intestine.³⁴ It has a pH optimum of 6.0 and molecular weight of 280,000. While lactose is not absorbed intact as a disaccharide to any significant extent, its two monosaccharide hydrolysis products are absorbed from the small intestine

into the enterocyte via the sodium-dependent hexose transporter, SGLUT-1, and then across the enterocyte basolateral membrane, into the hepatic portal vein via another glucose transporter, GLUT-2.³⁵ After entering the liver, much of the galactose is converted into glycogen and perhaps some to glucose. The latter can then enter the general bloodstream.³⁶

The perfectly normal metabolic phenomenon of lactose intolerance is not to be confused with a different, and grossly abnormal, disorder, congenital LPH deficiency (CLD). This latter condition is a very rare recessively inherited autosomal mutation in the translated region of the LPH gene which results in LPH activity being very low or absent in the intestinal epithelium from birth.^{37,38} Only a few dozen cases have been documented, most of them in Finland.²⁰ CLD will not be considered further in this review.

LPH in normal human neonates appears very late in fetal life. At 23 weeks gestation the LPH activity in the proximal jejunum is about 10% of that of a neonate and rises to 30% between 26 and 34 weeks gestation.^{39,40} In lactose intolerant human subjects (by far the majority of the World's population), LPH activity decreases progressively after weaning, generally becoming very low by five years of age.⁴¹⁻⁴³ Thus most humans have negligible LPH levels by the time they reach adulthood.⁴⁴ This loss of intestinal LPH activity is genetically programmed. It is also irreversible,⁴⁵ as subsequent consumption of lactose will not re-induce LPH in the human intestine.³⁴ However, there is a minority of humans (the so-called "lactase persisters" or "lactose tolerant" individuals) who maintain high levels of LPH throughout adult life.⁴⁴ Low LPH activity in the small intestine is the primary cause of lactose intolerance.^{46,47} Individuals who are "lactose intolerant" do not have sufficient LPH to hydrolyse the lactose they consume, or might otherwise consume. When these subjects ingest lactose, it enters the colon unchanged, to become a substrate for fermentation by bacteria. A failure to ferment most of the lactose in the colon often results in osmotic diarrhea.⁴⁸

Lactose tolerance has arisen as a result of genetic (and consequently ethnic) selection. It is an outcome of the development in certain human population groups of the technologies of pastoralism,⁴⁹ and is a classic anthropological manifestation of a genetic trait that has been influenced by cultural factors.⁵⁰ The economic agricultural dynamics of a given region during the past few thousands or tens of thousands of years have strongly influenced the frequency of lactose tolerance or intolerance in that region's population. Pastoralism in the distant past generally developed as a consequence of population growth in which the resulting pressure on resources lead to intensive agriculture. Land ownership and its associated fierce territorialism, which are intrinsic to agriculture, meant that only in certain parts of the world was there enough marginal land for pastoralism. So to feed domestic herds adequately, generally large distances had to be covered, involving a high labour requirement with specialization: in other words, a nomadic pastoral lifestyle.⁵¹ In some areas of the world, pastoralism has been a dominant feature of the local economy for many thousands of years. These areas include parts of Northern Europe, NE Africa, and the regions occupied by the Fulani of Western Africa, the Khoi pastoralists of Southern Africa and Bedouins. In these areas, there was (and still is) selective genetic pressure in which those who were able to consume a large amount of milk were more fertile or survived for longer, and therefore reproduced more effectively, than those who could not consume much milk. In other areas of the world, in which pastoralism was of little or of almost no importance, this selective pressure was less or absent.^{50,52-55}

The genetics and inheritance of lactose tolerance and intolerance

An examination of the expression of individual LPH mRNA transcripts in lactose tolerant subjects led to the discovery that one allele of the LPH gene is expressed at much lower levels than the other in those subjects with intermediate LPH activities, which are

nevertheless sufficient to hydrolyze the lactose load during a lactose tolerance test.⁵⁶ This strongly suggests that such individuals are heterozygous, having one LPH allele which is down-regulated during childhood, but in whom the other persists.⁴⁵ It is clear from these studies that the sequence differences responsible for LPH persistence/non-persistence reside within a cis-acting dominant regulatory gene mutation associated with the LPH gene, accounting for intermediate values in lactose tolerant heterozygotes. Hence persistence of intestinal LPH is caused by a genetic polymorphism, similarly to the inheritability of traits like colour-blindness or ABO blood type. Family studies have suggested that lactose intolerance is inherited as an autosomal recessive trait, or to put it in the opposite way, lactose tolerance is an autosomal dominant trait.³⁴

A study of Finnish families established an association between lactose intolerance and a C/T single nucleotide polymorphism (SNP) -13910 bases upstream from the LPH gene locus. Subjects with adult type LPH deficiency were shown to be generally homozygous for the C allele. In addition to this C/T SNP, a G/A SNP -22018 bases upstream from the LPH gene locus was reported. However, this latter polymorphism was not completely correlated with the LPH persistence/non-persistence phenotype, suggesting that the G/A SNP may be associated with, but not directly causative of, its associated phenotype.⁵⁷ While the C/T -13910 and

G/A -22018 SNPs are prevalent in European populations, they are almost nonexistent among sub-Saharan African populations that showed high LPH persistence. These populations were found to have different SNPs, amongst which were G/C -14010, T/G -13915 and C/G -13907.⁵⁸ All of these closely-associated polymorphisms are found in exon 13 of the MCM6 (DNA replication licensing factor minichromosome maintenance complex component 6) gene. In addition, 4 SNPs were identified in sub-Saharan Africans and Saudis, in the same gene region, namely T/G -13915 and perhaps linked to it, an SNP in exon 17 of the MCM6 gene, T/C -3712⁵⁷. A study of the prevalence of the C/T -13910 and G/A -22018 SNPs in 20 Brazilian subjects with (n=10) and without (n=10) lactose intolerance, diagnosed by the hydrogen breath test (HBT) has suggested the likelihood of there being additional SNPs in that population that could also lead to LPH persistence.⁵⁹ Figure 1 summarises the loci and polymorphisms associated with LPH persistence related to intron 13 of the MCM6 gene, that have been identified in three studies to date. The fact that six polymorphisms associated with LPH persistence have been characterised in these studies suggests that there may be others that have not yet been recognized. There is currently no information about genetic polymorphisms in the MCM6 gene in any of the many ethnic groups in Malaysia.

This effect would be expected to enhance the symptoms of mal-digestion⁶⁹ and to result in an increase in the load of lactose entering the colon. The consequence will be diarrhea, unless much of the lactose in the colon is fermented by anaerobic microorganisms.

The relationship between lactose load and symptoms of lactose intolerance in mal-digesters

There is a wide variation among lactose intolerant subjects with respect to the load vs. symptom responses.³⁴ Thus, it is hard to recommend a single threshold for lactose ingestion that is appropriate for all lactose mal-digesters. Some subjects complain of getting symptoms after consuming less than 6 g of lactose.²⁰ However, lactose-free control milk apparently induces symptoms in as many subjects as milk containing 7 g of lactose,⁷⁰ suggesting that psychosomatic symptoms may cloud the issue. Nevertheless, for any particular lactose intolerant individual, the clinical symptoms arising from lactose ingestion are clearly related to the dose. Many intolerant subjects can tolerate up to 12 g of lactose with no apparent symptoms, if lactose is taken as milk and with other foods.⁷¹ As the lactose concentration in commercially available cows' milk is approximately 50 g/L, this corresponds to 240 mL of milk, or around one standard cupful. One third to half of lactose intolerant individuals experience symptoms after ingesting this amount of milk.⁷² A single dose of 24 g of lactose (around 500 mL of milk) usually leads to appreciable symptoms, but can often be tolerated if consumed over the course of the day and with other foods.²⁰ Consumption of 50 g of lactose per day (approximately equal to the lactose in 1 L of milk) causes obvious (and frequently severe) symptoms in the majority of lactose mal-digesters.⁷³⁻⁷⁷ On the other hand, there is a small percentage of lactose intolerant subjects who remain apparently symptom-free even after ingestion of large amounts of lactose.⁷⁸ This is our observation also.

Gut flora and its effect on lactose fermentation in lactose intolerant subjects.

The colon is a complex and dynamic microbial

ecosystem with high densities of living bacteria (10^{11} or 10^{12} cells/g of luminal content). There are 300-500 different species of bacteria in the intestine.⁷⁹ The initial colonization of the gastrointestinal tract starts almost immediately after vaginal delivery⁸⁰ and affects the final composition of the permanent flora in adults.⁸¹ The predominant bacteria in the colon are from the genera *Bacteroides*, *Bifidobacterium*, *Eubacterium*, *Clostridium*, *Peptococcus*, *Peptostreptococcus*, and *Rumenococcus*.^{80,82} Subdominant genera include facultative anaerobes such as *Escherichia*, *Enterobacter*, *Enterococcus*, *Klebsiella*, *Lactobacillus*, *Proteus* and others. The species of bacteria found in each individual are distinct and vary greatly⁸³ accounting in part for the wide range of responsiveness to colonic lactose. An individual's colonic microflora will also change as a result of factors encountered later in life such as exposure to specific antibiotics and dietary factors. Thus repeated exposure of constipated patients to lactulose results in diminished breath hydrogen excretion,⁸⁴ due to the colonic proliferation of organisms such as *Bifidobacterium* spp. that can ferment lactulose (and lactose) via non-hydrogen-releasing pathways.

The fermentation of non-digestible carbohydrates (including large polysaccharides such as pectins, gums, cellulose, hemicelluloses, and resistant starches, some oligosaccharides, unabsorbed sugars, such as lactose, lactulose, stachyose and raffinose) particularly in the caecum and right colon, is very intense.^{82,85-88} These are converted to organic acids such as lactate, succinate, acetate, propionate and butyrate, with hydrogen gas and carbon dioxide gas as additional end-products. These acids stimulate bacterial growth but also provide appreciable sources of energy for human nutrition. Acetate is formed by many of the bacterial genera, and 60-75% of acetate is voided into human faeces.⁸⁹ However, propionate and butyrate have beneficial nutritional effects: propionate is largely metabolized in the liver and is gluconeogenic and inhibits lipogenesis; while butyrate is a major energy source for colonocytes, and may prevent colitis and colorectal cancer.^{90,91}

Adaptation to lactose consumption

While LPH undergoes an irreversible reduction in its activity after the neonatal period, the “induction or adaptative hypothesis” suggested that the presence of lactose in the diet could influence LPH activity and result in its persistence beyond the age of weaning.⁹² Evidence to support this hypothesis arose largely from animal studies in which very high-lactose diets were fed to rats for periods up to ten weeks, resulting in statistically significant increases in LPH activity.^{93,94} Studies in newborn rabbits⁹⁵ showed similar results. This induction phenomenon is now discounted for humans, but that does not mean that other forms of adaptation to a diet rich in lactose cannot occur. Continuous dietary intake of lactose reduces the severity of gastrointestinal symptoms in many lactose intolerant individuals.^{96,97} However, this effect is not due to a significant dietary-induced enhancement of LPH activity⁴⁴; the prevalence of lactose malabsorption is independent of the extent of milk consumption.^{60,98,99} In a recent study, 21 African-American adolescent girls consumed a dairy-rich diet of 33 g lactose per day for 21 days. For those who were lactose intolerant, both the symptoms of malabsorption and breath hydrogen excretion decreased significantly between the beginning and the end of the study,¹⁰⁰ suggesting that colonic adaptation to the high-lactose diet was occurring, possibly due to increased colonic acidity and changes in colonic flora.^{24,101} Continuous consumption of lactose for 6 days has been found to reduce fecal *Bacteroides* spp. and *Clostridium perfringens*, but to increase *Lactobacilli*, *Enterococci*, *Candida* ssp., and *Staphylococci*, resulting in an increase in fecal formic acid and valeric acid.¹⁰² This has been shown to be accompanied by an increase in microbial β -galactosidase activity.^{97,103,104} Hence, with repeated exposure to lactose in the colon, the colonic flora can change and adapt quickly to lactose, becoming capable of metabolizing increasing amounts.¹⁰⁵ Lactose-intolerant Nigerians who were introduced to ice cream containing lactose have reported initial symptoms that disappeared after several months of consumption.¹⁰⁶

Such “colonic bacterial adaptation” has also been observed in animals. Diarrhea was found to cease over time after continuous feeding of a diet containing 17% lactose to rats.¹⁰⁷ Hence the available evidence indicates that “colonic adaptation” to substantial levels of lactose in the diet can occur. The resulting changes in the flora of the large intestine include an enrichment of microbes with enhanced β -galactosidase activity, such as *Bifidobacteria* and other lactic acid bacteria, which can metabolize lactose without producing hydrogen and/or methane¹⁰⁶ to provide more energy-rich metabolic products than otherwise.

Dietary avoidance of the symptoms of lactose intolerance amongst the majority of Malaysians

The first and most crucial point that should be made is that at present, Malaysians simply do not consume sufficient milk and milk products throughout life to ensure maximum bone health in advanced age. If this situation does not change, the consequence is that, with the population living to an increasing age, the problems of osteoporosis in elderly women (and, to a lesser extent, men) will increase substantially in the future, and with it, attendant morbidity. One factor inhibiting the consumption of milk is the relatively high cost of dairy items. However, with increasing affluence in the future, this factor is likely to become of lesser significance, at least for much of the Malaysian urban population. To an extent which has not been satisfactorily investigated in Malaysia, the almost universal prevalence of lactose non-persistence is likely to be a second important factor inhibiting the consumption of milk and dairy products, as has been described elsewhere.^{2,3} Hence strategies to minimize the potentially adverse effects of lactose intolerance in Malaysia on consumption of dairy products are of importance.

Several options are available for the management of lactose intolerance in Malaysia. The first is to exclude lactose from the diet, as appears to have been the practice of many Malaysians. However, as we have seen, most lactose intolerant people can tolerate a

significant amount of milk without developing any signs or symptoms. Thus, totally eliminating lactose from the diet in these individuals is not necessary⁴⁴ and certainly undesirable.

As alternatives to reducing or eliminating lactose-containing foods, there are several other strategies that will permit lactose-intolerant individuals to consume dairy products. For instance, consumption of whole milk¹⁰⁸ or chocolate milk¹⁰⁹ rather than skim milk, and consuming milk with meals^{110,111} may reduce the resulting symptoms. In addition, fermented milk products such as aged cheeses (e.g. Cheddar and Swiss) usually contain significantly less lactose than the milk from which they were manufactured, and may not therefore induce the symptoms of intolerance. Furthermore, pre-hydrolyzed milk that has been treated with β -galactosidase derived from microorganisms is available from some dairy manufacturing companies. Moreover the addition of this enzyme to milk improves its absorption and tolerance.¹¹²⁻¹¹⁴ Two β -galactosidases have been produced commercially, one from the yeast *Kluyveromyces lactis* and another from the fungus *Aspergillus oryza*.¹¹⁵

Yogurt is now given much attention as a suitable alternative to milk for individuals who are lactose intolerant. Ingestion of 18 g of lactose in yogurt has been found to result in one third of the breath hydrogen response compared with the same amount of lactose in milk or water, and it also improves signs and symptoms associated with intolerance.¹¹⁶ Yogurt is made by inoculating milk with *Lactobacillus bulgaricus* or *Streptococcus thermophilus*. These organisms consume some of the lactose, while surviving gastric digestion, to become active at the temperature and pH of the duodenum.⁴⁴ Hence they reduce the level of lactose while contributing to its hydrolysis in the gastrointestinal tract.

Finally, while our own studies have provided evidence that lactose intolerant Malaysians do not become tolerant by consuming milk regularly, there was some indication

that colonic adaptation could occur, presumably by the mechanisms discussed above. So frequent exposure to milk products is likely to reduce the severity of any residual symptoms in a significant proportion (but certainly not all) of Malaysian consumers.

Conclusions

Our advice to the vast majority of Malaysians who are lactose-intolerant is as follows:

Some may get severe symptoms of maldigestion when consuming milk products, but the majority may not realise that they are lactose-intolerant, and those should consume more milk and its products.

1. In general a cup of milk should be well-tolerated by most non-persisters.
2. Milk should preferably be consumed with other foods, to slow the intestinal transit time for lactose.
3. The lactose in yogurt containing active bacterial cultures is better digested than the lactose in milk.
4. β -galactosidase supplements (pills, capsules) or lactose-hydrolyzed milk are good alternatives to unprocessed milk for those affected severely by the symptoms of lactose intolerance.
5. For many individuals, the repeated daily consumption of foods which contain lactose (milk and/or dairy products) may increase the fermentation ability of the bacteria in the colon to generate nutritious energy-rich products from the lactose therein.

Acknowledgements

I would like to thank my previous 4th Year Research Project Student from the School of Nutrition and Food Science, Universiti Malaysia Sabah, Ms Min Yen Tan, and her co-supervisor, my friend, Ms Yasmin Beng Ooi, of the same School, for their substantial contributions to the ideas, results and literature review herein. Without the help of both of these collaborators, this paper could not have been produced.

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