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Lactose Intolerance: An Overview of the Facts and Their Implications

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Abstract

Lactose intolerance is often blamed for the symptoms such as bloating, diarrhea, gas, abdominal pain, and nausea, that ail many people. Patients often do not seek proper diagnosis from a physician and create their own treatment plans, severely restricting lactose intake, without professional guidance. Even those who do seek the care of a physician find that diagnosis is complicated by less-than ideal testing and confusion due to the symptoms common to many other conditions. The misconceptions and inability to confirm a diagnosis of lactose intolerance can cause nutrient deficiencies in these patients, as well as begin a pattern of unnecessary lactose elimination that can continue for generations. The truth is that lactose restriction does help ease these symptoms regardless of their true cause, but complete elimination is not necessary in most cases. Each individual is unique and must experiment with their own tolerance to determine the level of lactose their body can digest. This paper aims to help those who might suffer from lactose intolerance understand the condition and maintain a healthy diet and lifestyle.

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Introduction

Digestion is an important process that is constantly taking place in the human body. However, it is a very complicated system that is both affected by and affects many factors of health and well-being. Sometimes, the digestive system doesn't work the way it is supposed to. While much is known about the digestive system and complications that can take place in the process, there are still many unknown aspects. One such topic is Lactose Intolerance, the phenomenon of gastrointestinal symptoms caused by lactase deficiency and the reduced ability to digest lactose. Despite the high prevalence of lactose intolerance in many parts of the world, there are still many components of the condition that are not well understood. A standardized method of testing for lactose intolerance has not yet been developed and many individuals self-diagnose without the proper information. Every individual experiences varying tolerance for lactose and severity of symptoms, further complicating the diagnosis and treatment. Currently, the only truly satisfying treatment is restricting lactose from the diet. Yet, complete avoidance of lactose is not necessary and can lead to deficits of important nutrients. It is clear that more work must be done to properly inform patients about lactose intolerance.

Background

Lactose

Lactose, also called milk sugar, is the main carbohydrate found in all mammalian milk except that of the sea lion (1, 5, 6, 8, 9, 11, 12). The disaccharide is composed of the

monosaccharides glucose and galactose (5, 11). It makes up about 7.2 g for every 100 mL of human milk and 4.7 g of cow's milk (7). Lactose is only about 1/6 as sweet as sucrose and less than half as sweet as glucose (8, 14). Lactose provides an excellent source of energy at a time of rapid growth and development during infancy (7). However, it has no special nutritional importance for adults (14).

Lactose has many purposes in food. Because it cannot be metabolized by yeast and it is not very sweet, it can be added to many foods and drinks (8). Lactose participates in the Maillard browning reaction, which results in a browner crust. Therefore, it is used as a browning agent in bread and cake mixes and is added to processed meats and sausages (8). It can also be added to soft drinks, lagers, breakfast drinks, powders, and sliming products (8). The gritty, hard crystals can easily be compacted, making lactose a good filler in pharmaceutical products as well. Annually, about 300 million kilograms of lactose are produced (8).

Digestion

Carbohydrates, which are ingested as starch (long polysaccharides of glucose) or sugars, account for about 50% of the calories in the average American diet (4). Carbohydrates must be digested to break down starch and sugars into monosaccharide units that can be absorbed for energy and nutrients. Cooking foods can be considered the start of this process because heat softens the tough, fibrous tissues of vegetables, fruits, and grains (2). When starch granules are heated, they swell and absorb water, making them easier for our bodies to digest.

In the body, the digestion of carbohydrates begins in the mouth. Salivary amylase, an enzyme in saliva, breaks the bonds between adjacent glucose molecules as the food is chewed (2, 4). However, carbohydrates are not sufficiently digested in the mouth because most people

do not chew their food long enough. The digestion of carbohydrates stops when the food is swallowed. When the food, now called a bolus, enters the stomach, the salivary amylase is inactivated by the acidic environment. Carbohydrate digestion does not continue until the bolus enters the small intestine where pancreatic amylase and dextrinase carry on the process (2).

The digestion of carbohydrates mainly takes place in the duodenum, the first portion of the small intestine (4). Here, pancreatic amylase cleaves the starches into maltose, maltriose, and oligosaccharides (short, branched chains of glucose molecules) which are hydrolyzed by brush border enzymes on the microvilli of the epithelium of the small intestine (4). Specialized enzymes in the absorptive cells of the small intestine break down specific disaccharides into their monosaccharide units. Maltase acts on maltose, a product of starch breakdown, to produce two glucose units. Sucrose, which is also known as table sugar, is acted upon by sucrase resulting in one glucose and one fructose molecule. Lastly, lactose is digested by lactase to produce glucose and galactose (8). These monosaccharides are absorbed into the epithelial cells by secondary active transport and glucose is further absorbed, by facilitated diffusion, into the capillaries (4). Dietary fibers and some starch from whole grains are indigestible carbohydrates which cannot be broken down in the small intestine. They pass to the large intestine where bacteria ferment them into acids and gases (2).

Lactase

The specific enzyme responsible for hydrolyzing lactose is B-galactosidase or lactase-phlorizin hydrolase, or more commonly known as lactase (5, 14). Lactase is found in the brush border of the villus epithelial cells in the small intestine (3, 13). It is most concentrated in the

jejunum, the middle portion of the small intestine (14). Located on the apical surface (the tips) of the villi, lactase is more susceptible to damage from intestinal diseases than enzymes located deeper in the brush border (7, 14). The enzyme has two active sites: one hydrolyzes lactose and the other phlorizin and other dietary glycolipids (7). Lactase is primarily responsible for breaking lactose into its monosaccharide components glucose and galactose, which can then be absorbed into the bloodstream (7, 9, 11-13). Glucose can then be utilized as a source of energy, while galactose becomes a component of glycolipids and glycoproteins (7).

Lactase activity can first be detected around the eighth week of gestation and peaks at the time of birth (7). Infants continue to produce high amounts of lactase as their main source of nutrition and energy comes from milk during this important time of growth and development (6, 7, 12). After weaning, around age 2-3 or as late as age 5, a genetically-programmed decrease in lactase activity begins (3, 9, 11, 12). Most humans lose 90-95% of the lactase levels from birth which then stabilize between ages 5 and 10 (9, 13). The mechanism responsible for this loss of lactase is unknown, but decreasing lactase levels after weaning is a characteristic of all mammals (8). For most adults, lactase activity does not seem to continue to decline with age, but about 30% of humans do continue to lose lactase activity in adulthood (7, 14). It is not fully understood whether continued use of dairy after weaning impacts the retention or decline of lactase activity (13).

Terminology

In order to begin understanding lactose intolerance, one first must learn the terminology surrounding lactose digestion and lactase activity. The usual reduction of lactase activity in the brush-border enzymes of the small intestine relative to that in infants is called

lactase deficiency or *lactase non-persistence* (11). Conversely, the continuation of high levels of lactase activity into adulthood is called *lactase persistence. Lactose malabsorption* is the physiological problem where a substantial amount of ingested lactose is not absorbed in the intestine due to a deficiency of the lactase enzyme (11, 12). In other words, the amount of ingested lactose exceeds the ability of lactase to hydrolyze it (5). Usually, lactose malabsorption is caused by lactase deficiency (non-persistence) or impaired activity of the enzyme, but can also occur due to other intestinal pathologies (9). However, lactose malabsorption does not necessarily indicate that the individual will have symptoms (12). When lactose malabsorption does cause gastrointestinal symptoms, it is called *lactose intolerance* (8, 11, 12). More formally, lactose intolerance is a clinical condition characterized by the "onset of gastrointestinal symptoms following blinded, single-dose challenge of ingested lactose by an individual with lactose malabsorption, which are not observed when the person ingests an indistinguishable placebo" (5, 9). Therefore, the condition of lactose intolerance requires not only evidence of lactase deficiency but also the development of symptoms in a blind placebo-controlled lactose challenge (9). It is important to note that these two conditions are not the same. Lactose malabsorption can be subjectively verified through clinical tests, but generally, does not require the attention of a physician. The symptoms required to be considered lactose intolerant can be caused by many factors including diet, oro-cecal transit time, variability of gut flora and psychological factors (9). Lactase deficiency and lactose malabsorption are only one potential cause of the condition. Thus, the prevalence of lactose malabsorption is greater than that of lactose intolerance because not all patients with lactose malabsorption experience symptoms (11).

Prevalence

Somewhere between two-thirds and three-fourths of the world's population experience lactose intolerance (1, 5, 7, 8, 14). In North America about 25% of adults show decreased lactose digestion. Worldwide, the prevalence of lactose intolerance is above 50% in South America, Asia, and Africa and is almost 100% in some Asian countries (14). Northern Europeans, North Americans, and Australians have the lowest rates of lactose intolerance (7). Sufficient data is not available to determine the prevalence of lactose intolerance in the United States (12). It is known that European Americans have the lowest prevalence of lactose intolerance while African-, Hispanic-, Asian-Americans, and American Indians have a higher occurrence (12). Some estimates for prevalence in the U.S. are about 15% for whites, 50-80% for Mexican-Americans, and 60-80% for African Americans, and near 100% for Asian-Americans and American Indians (5, 14). Gender does not seem to have any effect on the prevalence of lactose intolerance (14). Northwestern Europeans, particularly Swedes and Danes, show the highest prevalence of lactase persistence or the ability to digest lactose with 80-95% lactase persistence (3). Persistence is also frequent in some nomadic Afro-Arabian desert regions where pastoral lifestyles were more common (3). The prevalence of lactase persistence is 20-40% in India, about 30% in Mexico, and about 30% in Africa (9). The lowest prevalence of lactase persistence occurs in Asiatic populations (less than 10%) (6, 9). The high prevalence of lactose intolerance around the world leads to the belief that non-persistence is the ancestral type and lactase persistence is the mutated condition (7). Humans are unique in that some racial types have lactase persistence into adult life as a result of selective pressures allowing the mutant gene to continue (3).

History

Natural selection has played a major role in the variable frequency of lactase persistence amongst different populations of humans (6). The higher lactase persistence in northern Europeans could be geographically linked to the prominence of dairy farming in this area beginning around 10,000 years ago. This theory, termed the "culture-historical hypothesis", claims that genetic lactase persistence was rare prior to the introduction of dairy farming (7). It argues that mammalian milk became an important component of the diet during times of poor harvest (7, 15). Milk provided a clean source of fluid, energy, and nutrients, leading to a strong selective advantage and pressure for those who could digest it (9). Thus, lactase persistence developed independently several times in human evolution as different regions of the world started farming about 10,000 years ago (9). Now, in areas with long traditions of dairy farming, lactase persistence is more common (14). The opposing "reverse cause" theory claims that those with a pre-existing lactase persistence adopted dairy farming and consumption (7). However, this theory has less supporting evidence. With the expansion of the dairy industry and migration of populations, many lactase deficient adults live in societies where lactose-containing foods are a regular part of the diet (3). Although most research suggests that the mutation causing lactase persistence only arose in humans, not much is known about lactose intolerance in other higher primates. Another theory is that lactose tolerance evolved to enable primates to delay weaning, allowing them to protect their young for longer and have longer periods between births (6).

The first descriptions of lactose intolerance came from Hippocrates around 400 B.C., but the clinical symptoms have only been recognized in the last 50 years (7). The first examples of

lactase non-persistence were considered an abnormal trait, but it was soon recognized that this supposed abnormality was the most frequent trait worldwide (6). For this reason, some experts do not regard lactose intolerance as a clinically serious condition. Dr. Michael D. Levitt of the Minneapolis Veterans Affairs Medical Center said that most people with an inactive lactase gene rarely have problems unless they drink large amounts of milk (15). He believes that most people who think they have lactose intolerance actually have irritable bowel syndrome and would like to find a cause, including that the condition is "mostly an American phenomenon, and the rest of the world is not much interested in it," (15).

Causes

The most frequent cause of lactose malabsorption is lactase non-persistence (5). This condition is termed primary lactose intolerance and is characterized as the genetic reduction of lactase activity that occurs after weaning in almost all animals and humans (5, 13, 14). Activity normally drops to one-tenth or less of the peak levels during infancy by adulthood and the condition of lactase non-persistence continues throughout the lifespan (13, 14).

Secondary lactose intolerance is an acquired form of lactose intolerance in people with lactase persistence. Here, lactase deficiency is caused by an injury that damages the epithelium (lining) of the small intestine and leads to a reduction of the absorptive capacity for lactose or a down regulation of lactase expression (5, 9, 13). This may be due to small intestinal resections, gastroenteritis, celiac disease, severe diarrhea, infections, drugs, or Crohn's disease. In this case, lactose intolerance is reversed when the intestinal lining heals and lactase production normalizes (5, 7, 8, 14). Secondary lactose intolerance can affect people at any age, but is common in infancy (5).

Lastly, congenital lactase deficiency, called alactasia, is a rare condition where lactase is completely absent from the small intestine from birth (5, 7, 8). Congential lactose intolerance presents with severe symptoms at birth, such as diarrhea from first exposure to breast milk, and is characterized by failure to thrive (7, 9). It can be a deadly condition for newborns due to the dehydration and electrolyte loss associated with diarrhea (5). The only treatment for this rare form of lactose intolerance is the complete avoidance of lactose (7). Little is known about the cause of this condition as only a few dozen cases have been documented in the world (14).

Symptoms

The symptoms of lactose intolerance can lead to significant discomfort, often decreasing quality of life and leading to absence from school and work (5). The gastrointestinal symptoms include abdominal pain, bloating, flatulence, borborgymi, nausea, and diarrhea or loose stools when sufficient lactose is consumed (3, 9, 11, 13, 14). Bloating and gas are caused by the fermentation of unabsorbed lactose in the colon (large intestine) (13). The gut flora (bacteria) of the large intestine metabolize the lactose by cleaving it into short-chain fatty acids and gases such as hydrogen, carbon dioxide, and methane as a salvage pathway for lactose digestion (5, 9). Undigested lactose also draws water into the large intestine to maintain osmotic equilibrium, which causes diarrhea or loose stools (5, 8, 9, 13). The dilation of the small intestine accelerates transit which further increases maldigestion of lactose, and other nutrients, because of the reduction of contact time between lactose and enzymes (14). Other symptoms of lactose intolerance may include vomiting, constipation, headaches, lightheadedness, loss of concentration, fatigue, and ulcers (8, 9). The breakdown of lactose by bacteria in the colon generates hydrogen and other metabolites that can be toxins that act on

ionic signaling pathways in the nervous system, heart, muscles, and immune system, causing a variety of seemingly unrelated symptoms (8).

Although many people have decreased lactase production in adulthood, they can usually tolerate some lactose without presenting symptoms. First, this is because although the activity of lactase is decreased, it does not stop completely. The lactase available does digest some lactose. As well, bacteria or flora in the large intestine break down the lactose. It has been found that most patients with self-reported lactose intolerance can ingest about 12 grams of lactose, the amount in about 1-2 cups of milk, without the presence of symptoms and even 15-18 g if eaten with other foods (5, 9, 11-13). A dose of 24 g usually induces substantial symptoms, but some patients rarely experience symptoms at all (11, 14). Generally, fermented dairy products are better tolerated because the lactose in these foods has been converted into lactic acid during their production (1). Yogurt tends to cause fewer symptoms than milk for those who cannot digest lactose, along with hard cheeses (14). For instance, 1 kilogram of parmesan cheese has about the same amount of lactose as a glass of milk, so a spoonful on top of a plate of pasta isn't likely to cause symptoms (8). Butter has only a trace amount of lactose and is also generally eaten in small quantities, so it should not cause symptoms alone (8). The low amounts of lactose in baked products and added to margarines or sausages can usually be tolerated (3).

Because many factors influence the development and severity of symptoms, diagnosis and treatment of lactose intolerance becomes complicated. The development of symptoms after the ingestion of lactose is influenced by many factors such as lactase activity, gastric emptying rates, fecal bacterial metabolites, colonic mucosal absorptive capacity, and intestinal

transit time (12). The severity of the symptoms experienced depends on the amount of lactose ingested above the individual's lactase threshold (5, 11, 13). Individuals differ in the intensity of symptoms experienced because of differences in pain perception and the psychological effect of pain and social discomfort (12). The tolerable load of lactose may vary depending on whether other nutrients are consumed along with the lactose (11). Some lactose maldigesters experience symptoms even when given a placebo, while others remain symptom-free after ingesting large amounts of lactose (14). It is nearly impossible to determine a minimum lactose tolerance because each case is unique.

Diagnosis

As previously mentioned, the diagnosis of lactose intolerance requires both evidence of lactase deficiency and the development of symptoms from lactose ingestion. While lactose malabsorption is necessary, it is not enough evidence to diagnose the patient as lactose intolerant (9). Dietary history alone is also not a reliable means to confirm or deny the diagnosis of lactose intolerance due to varying patient reports and the potential of a placebo effect (5). In population-based studies, between 9.5% and 25% of the community self-reported food intolerances. When tested in a controlled-blinded study, only 25.4% of these patients reacted to the offending food (9). In addition, 59% of subjects with self-reported severe lactose intolerance with lactose malabsorption had no significant effects in a placebo-controlled challenge (9). Diagnosis can be very subjective because it is usually based on self-reported symptoms. Many individuals ascribe symptoms of diverse intestinal disorders to lactose intolerance without undergoing testing and the misconception often becomes intergenerational (12). Symptoms are also highly susceptible to the placebo effect, where the

patient may believe they have lactose intolerance and present with symptoms even though they cannot be attributed to any lactase deficiency or lactose malabsorption. Patients who present with the symptoms of lactose intolerance without the presence of lactase deficiency are said to have “functional lactose intolerance” because the condition still impacts the patient.

There are currently various options for diagnosing lactose malabsorption, including direct or indirect methods, such as lactose-tolerance tests, breath hydrogen tests, and biopsy assays (3). Direct measurement includes an analysis of the mucosal disaccharides (14). An assay of the lactase from a small-intestine biopsy or mucosal biopsies of the duodenum can directly determine the lactase activity (6, 12). This biopsy may also help to exclude other conditions that cause secondary lactose malabsportion (9). However, this technique is limited by the “patchy expression” of lactase and the invasiveness of the test (9). This method is more reliable than indirect methods, but is not as practical in neither clinical nor research settings. There is currently no genetic test for lactose intolerance (8). Genetics tests can help to identify lactase non-persistence, but currently only the most common polymorphism for lactase non-persistence in Caucasian patients (9, 12). Further research is needed to determine the specific polymorphisms in African, Arabic, or Asian subpopulations in order to make genetic testing useful for all patients. Additionally genetic tests will be negative for patients whose lactase deficiency is caused by a secondary factor (9).

Indirect methods to determine lactose malabsorption include lactose tolerance tests or breath hydrogen tests. In a lactose tolerance test, lactose digestion is measured through increases in blood glucose after the intake of a 20 to 50 gram lactose load or 1-1.5 g/kg of body weight, which is the equivalent to that found in about one liter of milk (9, 13). A significant

increase in blood glucose indicates the successful digestion of lactose or high lactase activity. Therefore, a rise in blood glucose of 26 mg of glucose per deciliter of blood after 30 minutes would indicate high lactase activity or lactase persistence (5, 6). If blood glucose increases less than 20 mg/dL above fasting level and symptoms occur, the test is positive for lactose intolerance (13). However, this test may be confused if the patient's blood sugar is also affected by impaired glucose tolerance or diabetes (9). False positives are common because of the normal insulin response to carbohydrates (5). This test is generally not sensitive enough to be useful.

Diagnosis of lactose intolerance is most commonly done by a lactose hydrogen breath test (7). Undigested lactose is fermented and produces fatty acids and gases when it reaches the colon, including hydrogen that is later excreted in the breath. Therefore, elevated hydrogen levels in the breath indicate lactase non-persistence (12). This test begins with the ingestion of some amount of lactose- either 50 g, 2 g per kg of body weight, or 25 g after fasting overnight, or even just 12 g- followed by measuring breath hydrogen levels every thirty minutes over the next three to six hours (5, 8, 9, 12, 13). An increase in breath hydrogen levels greater than 20 parts per million over baseline generally indicates lactose intolerance (5, 8, 13). However, many factors can affect the results of this test including sleep, exercise, aspirin use, and smoking (13). A false-negative may be obtained through this test due to variations in intestinal bacteria and flora and other factors affecting intestinal motility (Hayman 2006, 9). This method is currently considered the most cost-effective, non-invasive, and reliable way to measure lactose maldigestion (7). In both of these methods, some control subjects with no history of gastrointestinal problems exhibit symptoms after the intake of 50 grams of lactose (8). As well,

these tests lack a component that records the subjects' symptoms and can cause severe symptoms lasting several days (8).

It is quite evident that the methods of diagnosing lactose intolerance are inadequate to efficiently and effectively diagnose patients. A few changes would help improve this system and ultimately help patients be able to manage their symptoms to lead the most normal life possible. Blinded testing for lactose intolerance, which is testing without the patient's knowledge of their intake during the test, would reduce the effects of a placebo response (9). This would increase the specificity of the diagnosis and, therefore, guide the physician to a more reasonable dietary management of the symptoms. A suitable placebo must be developed before this can be implemented. Currently, there is no substitute to lactose that a subject cannot guess whether they are taking lactose are not (8). Additionally, multiple-dosage tests would eliminate doubts of the diagnosis and help the patient to better define their threshold for lactose (9). Many patients underestimate how much lactose they can safely ingest, leading them to overly reduce their lactose intake. Currently, placebo-controlled and multiple-dose testing are not used outside of research because they difficult to implement in clinical practice (9). One solution to this could be that, after the diagnosis of lactase deficiency in the laboratory, blinded testing at multiple doses would be completed at home.

Diagnosis is also confused because gastrointestinal symptoms are very common. Milk and dairy foods are often blamed for their occurrence even though they can be caused by many factors independent of lactose intake (14). Lactose intolerance and irritable bowel syndrome (IBS) exhibit many of the same symptoms, so both conditions should be considered by the physician when making a diagnosis (3, 13, 14). At least 25% of patients with IBS have lactose

malabsorption as well, so lactose restriction may result in improvement of symptoms in both groups anyways (13).

Treatment

Lactose Abstinence

The main goal of treating lactose intolerance is to manage the symptoms rather than reducing lactose malabsorption or increasing lactase activity (9). Patients should understand that they are not allergic to milk, dairy products, or dairy foods (13). Therefore, most patients do not require a totally lactose-free diet and should be reassured that complete lactose exclusion is unnecessary (3, 13). Careful diagnosis should be made before lactose is restricted (14). However, a diet low in lactose is the only truly satisfactory treatment for lactose intolerance at this time (3).

The most common strategy to treating lactose intolerance is to limit consumption of foods containing lactose or manipulate the circumstances where lactose is ingested in order to prevent symptoms. A reduced-lactose diet, limiting milk and milk-containing products, can decrease gastrointestinal symptoms in patients (12). About 85% of patients with lactose malabsorption reported improvement in their symptoms after restricting lactose. Every patient has a unique tolerance to lactose based on many factors, including amount of lactose ingested, lactase activity, colonic flora, ingestion of other food with lactose, gastrointestinal transit time, and individual sensitivity to symptoms (12, 14). Therefore, patients may need to take some time to understand their own abilities and limits in terms of reducing lactose in their diet individually.

While each patient is unique, some trends exist in circumstances where lactose is better tolerated which patients may want to experiment with when figuring out how their body handles lactose. Many studies have found that lactose is better tolerated in small amounts throughout the day than a single, large dosage (5, 11). Lactose is also better tolerated when ingested along with other foods than separately (11, 14). In general, delaying gastric emptying, or slowing transit time, allows for better digestion of lactose because the limited amount of the lactase enzyme has greater opportunity to come in contact with the ingested lactose. Therefore, full fat milk is better tolerated than skim milk, cold foods are better tolerated than hot, and solid foods are better tolerated than liquids (14). For example, yogurt, being a more solid food, may be a better option than milk, a liquid, even if they're measured to contain the same amount of lactose (14).

Enzyme Replacement

Another strategy used to avoid the symptoms of lactose intolerance is lactase enzyme replacement (5). Lactase supplements may be taken to aid in the digestion of lactose or the patient may choose to consume products that have been treated with enzymes to breakdown the lactose before digestion. There are many pharmaceutical preparations of lactase, such as DairyEase, that can help increase lactose digestion and prevent symptoms, even allowing patients to freely consume lactose (5, 13, 14). These supplements vary in effectiveness and it can be difficult to determine their most effective dosages (11, 13).

Consuming lactose-reduced milk and other dairy products is another option for patients. β- galactosidase, which is available in many health food stores, can be added to foods to reduce the amount of lactose in them (8). This method may change the taste of the product because

glucose and galactose, the components of lactose, are sweeter than the original disaccharide (9). Most supermarkets also sell low-lactose milk and dairy products, along with many non-dairy substitutes for these products such as soy, rice, or almond milk (8). Reduced-lactose dairy and nondairy alternatives are typically fortified with calcium, vitamin D, and other nutrients, making them good choices to replace dairy (5). However, they may be more expensive and less available (12).

Colonic Adaptation

Some patients may increase their tolerance to lactose by steadily increasing their dietary load of lactose over time or through routine ingestion of lactose (11-13). Lactase activity is not upregulated by lactose ingestion (9). That is, lactase activity will not be increased because of an increased demand for it by ingesting lactose. The repeated ingestion of lactose does help the intestinal flora adapt to better handle lactose digestion (9). However, studies have not found sufficient data to determine the effectiveness of colonic adaptation to treat lactose intolerance (11).

Nutrition

Many patients may choose to eliminate lactose from their diet, without making a proper diagnosis and treatment plan with their physician. Dairy, the main source of lactose, is also a source of many important nutrients, so any patient eating a lactose-free diet must also monitor these nutrients to ensure consumption of the recommended amounts. Dairy provides calcium, protein, magnesium, potassium, phosphorus, riboflavin, and vitamin A and D (especially when fortified) (12, 13). However, more studies are needed to confirm if those with lactose intolerance suffer from any important nutritional deficiencies and the presence of any risks

associated with lactose-free diets (5, 12). An overall nutritional eating plan should be recommended to patients who are concerned about lactose intolerance, with a focus on nutrients that may potentially by a reduced dairy or dairy-free diet (12).

The main concern in restricting dairy consumption is that it will result in the patient not meeting the daily recommended intake levels of calcium (5, 11). Calcium is necessary during childhood to reach optimal peak bone mass (12). Proper intake of calcium as a child determines risk for osteoporosis and fragility fractures later in adult life (12). Calcium is also necessary in adulthood to maintain bone density and reduce risk of fractures (12, 14). Meeting adequate intake of nutrients for skeletal health is especially important in adolescence, pregnancy and lactation, and older age (12). Many adults and children who avoid dairy products are not ingesting adequate amounts of calcium, vitamin D, and other essential nutrients (12). However, it is not certain whether lactose affects the efficiency of calcium absorption (5, 12). Regardless, it is important that people with lactase non-persistence seek calcium from alternative food sources or supplements (5, 9).

The current dietary reference intakes suggest a daily intake of calcium of 1000 mg per day for adults, 1300 mg for adolescents, and 1200 mg for those over 50 years of age (9, 10). Dairy foods, such as milk, yogurt, and cheese, provide the majority of calcium available in the United States food supply (10, 13). Patients must diligently seek calcium from other sources in order to achieve calcium intake of 1,000 to 1,500 mg per day (13). There are plenty of alternative foods to dairy that provide significant amounts of calcium. Some vegetables, such as kale and broccoli, are good sources of calcium, while others, such as spinach, provide calcium but have poor bioavailability (10). Grains contains low amounts of calcium (unless fortified) but

can contribute because people eat them frequently (10). A slice of whole-wheat bread contains only 30 mg of calcium and a flour tortilla has only 32 mg, so it is not realistic to rely on these as a significant source of calcium. Even more foods are fortified with calcium such as fruit juices and cereals (10). For example, eight ounces (one cup) of milk provides about 276 mg of calcium, while six ounces fortified orange juice contains about 261 mg and eight ounces of soymilk has 299 mg (10). Therefore, achieving the recommended daily intake of calcium is definitely doable without consuming dairy. Patients may also consider taking calcium supplements (13). Supplements are both inexpensive and convenient (10). About 43% of the United States population, and almost 70% of older women, utilize dietary supplements of calcium of an average of 330 mg/day (10).

Conclusion

Lactose intolerance is a condition afflicting many people in the world today. The symptoms of lactose intolerance, including abdominal pain, bloating, flatulence, borborgymi, nausea, and diarrhea, can be very uncomfortable and inhibit the patient from living a normal life. Although various methods have been used to diagnose it, lactose intolerance remains elusive due to complications posed by various symptoms of lactose intolerance, their similarity to those of other conditions, and their susceptibility to the placebo effect. If suspected, patients may use techniques such as lactose restriction, enzyme supplements, and colonic adaption, to manage their symptoms and return to a fairly normal lifestyle. Meanwhile, patients should be aware of nutritional deficiencies associated with their modified diets, especially of calcium. Patients should be conscientious of food alternatives to dairy that can provide calcium in order to maintain the recommended daily intake and may consider taking calcium supplements.

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Lactose-Free Recipe Guide

This guide provides simple recipes to help those who want to utilize a dairy or lactose-free diet in order to control their gastrointestinal symptoms. Some recipes focus on eliminating dairy from favorite recipes such as creamy pesto or chocolate mousse. Others emphasize ingredients that are rich sources of calcium, a nutrient commonly lacking in a dairy-free diet, such as kale, white beans, broccoli, and almonds. Each recipe features a nutrient analysis from www.supertracker.usda.gov that will help the user fit these recipes into their healthy diet and track important nutrients.

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Brown Sugar Almond Oatmeal with Apricots

Servings: 4

Ingredients

2 cup old-fashioned oats

3 ½ cup + 1 Tbsp. water

1 cup almonds

4 Tbsp. + 2 tsp. brown sugar

16 dried apricot halves, sliced

Steps

For the almonds:

1. Combine 3 Tbsp. brown sugar and 1 Tbsp. water in a microwave safe bowl.
2. Microwave for about 2 minutes until fully melted and thick.
3. Add almonds and fold to fully coat them.
4. Microwave for another two minutes.
5. Spread almonds onto a non-stick surface such as a parchment paper lined baking sheet.
6. Chill for at least an hour, ideally over night.
7. Chop almonds in a food processor.

For the oatmeal:

1. Prepare oatmeal according to the package's instructions.
2. Serve ¼ of warm oatmeal with ½ tsp. brown sugar, ¼ cup of almonds, and 4 sliced dried apricots.

Brown Sugar Almond Oatmeal with Apricots

Portions: 4

Food Groups	Amount Per Portion
Grains	2 ounce(s)
Whole Grains	2 ounce(s)
Refined Grains	0 ounce(s)
Vegetables	0 cup(s)
Dark Green	0 cup(s)
Red & Orange	0 cup(s)
Beans & Peas	0 cup(s)
Starchy	0 cup(s)
Other	0 cup(s)
Fruits	¼ cup(s)
Fruit Juice	0 cup(s)
Whole Fruit	¼ cup(s)
Dairy	0 cup(s)
Milk & Yogurt	0 cup(s)
Cheese	0 cup(s)
Protein Foods	3 ounce(s)
Seafood	0 ounce(s)
Meat, Poultry & Eggs	0 ounce(s)
Nuts, Seeds & Soy	3 ounce(s)
Oils	3 teaspoon
Limits	**Amount Per Portion**
Total Calories	482 Calories
Added Sugars	56 Calories
Saturated Fat	21 Calories
Nutrients	**Amount Per Portion**
Protein	14 g
Carbohydrate	56 g
Dietary Fiber	9 g
Total Sugars	25 g
Added Sugars	14 g
Total Fat	25 g
Saturated Fat	2 g
Monounsaturated Fat	15 g
Polyunsaturated Fat	7 g
Linoleic Acid	6 g

α-Linolenic Acid	0.0 g
Omega 3 - EPA	0 mg
Omega 3 - DHA	0 mg
Cholesterol	0 mg
Minerals	**Amount Per Portion**
Calcium	154 mg
Potassium	608 mg
Sodium	147 mg
Copper	589 µg
Iron	4 mg
Magnesium	171 mg
Phosphorus	359 mg
Selenium	13 µg
Zinc	3 mg
Vitamins	**Amount Per Portion**
Vitamin A	25 µg RAE
Vitamin B6	0.1 mg
Vitamin B12	0.0 µg
Vitamin C	0 mg
Vitamin D	0 µg
Vitamin E	11 mg AT
Vitamin K	1 µg
Folate	25 µg DFE
Thiamin	0.2 mg
Riboflavin	0.4 mg
Niacin	2 mg
Choline	39 mg
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Serving Tips

- Prepare enough for the whole week to save time in the mornings.
- Add cinnamon to almonds if desired.
- Substitute various dried fruits such as cranberries or dates or fresh fruits such as blueberries, raspberries, or blackberries.

Orange Cranberry Scones

Servings: 8

Ingredients

2 cups flour

1/3 cup sugar

1 Tbsp. baking powder

1 tsp. salt

1 cup almond milk

¼ cup orange juice with calcium and vitamin D

Steps

1. Preheat the oven to 425F.
2. Combine dry ingredients in a bowl.
3. Slowly stir in almond milk and orange juice.
4. Fold in dried cranberries.
5. Grease a baking sheet (may need two).
6. Scoop batter onto the baking sheet(s) with plenty of space between each, forming approximately eight scones.
7. Bake for 15-20 minutes total.
8. Before the last five minutes of baking, sprinkle sugar on top of each scone.
9. Serve warm.

Orange Cranberry Scones

Portions: 8

Food Groups	Amount Per Portion
Grains	2 ounce(s)
Whole Grains	0 ounce(s)
Refined Grains	2 ounce(s)
Vegetables	0 cup(s)
Dark Green	0 cup(s)
Red & Orange	0 cup(s)
Beans & Peas	0 cup(s)
Starchy	0 cup(s)
Other	0 cup(s)
Fruits	¼ cup(s)
Fruit Juice	0 cup(s)
Whole Fruit	0 cup(s)
Dairy	0 cup(s)
Milk & Yogurt	0 cup(s)
Cheese	0 cup(s)
Protein Foods	0 ounce(s)
Seafood	0 ounce(s)
Meat, Poultry & Eggs	0 ounce(s)
Nuts, Seeds & Soy	0 ounce(s)
Oils	0 teaspoon
Limits	**Amount Per Portion**
Total Calories	185 Calories
Added Sugars	54 Calories
Saturated Fat	1 Calories
Nutrients	**Amount Per Portion**
Protein	3 g
Carbohydrate	42 g
Dietary Fiber	1 g
Total Sugars	16 g
Added Sugars	14 g
Total Fat	1 g
Saturated Fat	0 g
Monounsaturated Fat	0 g
Polyunsaturated Fat	0 g
Linoleic Acid	0 g

α-Linolenic Acid	0.0 g
Omega 3 - EPA	0 mg
Omega 3 - DHA	0 mg
Cholesterol	0 mg
Minerals	**Amount Per Portion**
Calcium	175 mg
Potassium	66 mg
Sodium	494 mg
Copper	60 µg
Iron	2 mg
Magnesium	11 mg
Phosphorus	78 mg
Selenium	11 µg
Zinc	0 mg
Vitamins	**Amount Per Portion**
Vitamin A	19 µg RAE
Vitamin B6	0.0 mg
Vitamin B12	0.4 µg
Vitamin C	3 mg
Vitamin D	0 µg
Vitamin E	1 mg AT
Vitamin K	0 µg
Folate	93 µg DFE
Thiamin	0.3 mg
Riboflavin	0.2 mg
Niacin	2 mg
Choline	4 mg

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Serving Tips

- Serve warm.
- May make an orange glaze instead of sugar topping.

Kale Chips

Servings: 8

Ingredients

1 bunch of raw kale

Kosher salt (~1 tsp.)

Olive oil (~ 1 Tbsp.)

Steps

1. Preheat the oven to 300F.
2. Thoroughly wash and dry kale.
3. Tear kale leaves off of stems.
4. May tear kale into smaller pieces of desired size.
5. Spread kale onto an ungreased baking sheet.
6. Drizzle kale with olive oil and sprinkle with kosher salt.
7. Bake for about 10 minutes, rotate the pans and bake for another 15 minutes.
8. Remove from the oven and let cool for about 3 minutes, then store in a sealed plastic bag.

Kale Chips

Portions: 4

Food Groups	Amount Per Portion
Grains	0 ounce(s)
Whole Grains	0 ounce(s)
Refined Grains	0 ounce(s)
Vegetables	½ cup(s)
Dark Green	½ cup(s)
Red & Orange	0 cup(s)
Beans & Peas	0 cup(s)
Starchy	0 cup(s)
Other	0 cup(s)
Fruits	0 cup(s)
Fruit Juice	0 cup(s)
Whole Fruit	0 cup(s)
Dairy	0 cup(s)
Milk & Yogurt	0 cup(s)
Cheese	0 cup(s)
Protein Foods	0 ounce(s)
Seafood	0 ounce(s)
Meat, Poultry & Eggs	0 ounce(s)
Nuts, Seeds & Soy	0 ounce(s)
Oils	2 teaspoon
Limits	**Amount Per Portion**
Total Calories	93 Calories
Added Sugars	0 Calories
Saturated Fat	9 Calories
Nutrients	**Amount Per Portion**
Protein	3 g
Carbohydrate	6 g
Dietary Fiber	1 g
Total Sugars	0 g
Added Sugars	0 g
Total Fat	7 g
Saturated Fat	1 g
Monounsaturated Fat	5 g
Polyunsaturated Fat	1 g
Linoleic Acid	1 g

α-Linolenic Acid	0.2 g
Omega 3 - EPA	0 mg
Omega 3 - DHA	0 mg
Cholesterol	0 mg
Minerals	**Amount Per Portion**
Calcium	101 mg
Potassium	329 mg
Sodium	1188 mg
Copper	1005 µg
Iron	1 mg
Magnesium	32 mg
Phosphorus	62 mg
Selenium	1 µg
Zinc	0 mg
Vitamins	**Amount Per Portion**
Vitamin A	335 µg RAE
Vitamin B6	0.2 mg
Vitamin B12	0.0 µg
Vitamin C	80 mg
Vitamin D	0 µg
Vitamin E	2 mg AT
Vitamin K	476 µg
Folate	21 µg DFE
Thiamin	0.1 mg
Riboflavin	0.1 mg
Niacin	1 mg
Choline	0 mg
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Serving Tips

- Thoroughly dry kale after washing to avoid steaming it in the oven, causing soggy chips.
- Spread kale in a single layer so that it bakes evenly.
- Experiment with seasonings and dips to customize.
- Store in an air-tight container or bag to keep fresh.

Creamy Avocado Pesto

Servings: 4

Ingredients

½ cup pine nuts

2 cups fresh basil leaves (2.5 oz. package)

2 avocados

1 lemon, juiced

2 tsp. minced garlic

Olive oil (~ 2 Tbsp.)

Steps

1. In a food processor, chop pine nuts coarsely.
2. Add basil and garlic and continue to chop until fine.
3. Add avocado (cut in chunks or slices) and the lemon juice to the food processor; blend until smooth.
4. Add olive oil while blending until it achieves your desired consistency.

Creamy Avocado Pesto

Portions: 4

Food Groups	Amount Per Portion
Grains	0 ounce(s)
Whole Grains	0 ounce(s)
Refined Grains	0 ounce(s)
Vegetables	¾ cup(s)
Dark Green	0 cup(s)
Red & Orange	0 cup(s)
Beans & Peas	0 cup(s)
Starchy	0 cup(s)
Other	¾ cup(s)
Fruits	0 cup(s)
Fruit Juice	0 cup(s)
Whole Fruit	0 cup(s)
Dairy	0 cup(s)
Milk & Yogurt	0 cup(s)
Cheese	0 cup(s)
Protein Foods	1 ounce(s)
Seafood	0 ounce(s)
Meat, Poultry & Eggs	0 ounce(s)
Nuts, Seeds & Soy	1 ounce(s)
Oils	5 teaspoon
Limits	**Amount Per Portion**
Total Calories	289 Calories
Added Sugars	0 Calories
Saturated Fat	29 Calories
Nutrients	**Amount Per Portion**
Protein	4 g
Carbohydrate	10 g
Dietary Fiber	5 g
Total Sugars	1 g
Added Sugars	0 g
Total Fat	28 g
Saturated Fat	3 g
Monounsaturated Fat	15 g
Polyunsaturated Fat	8 g
Linoleic Acid	7 g

α-Linolenic Acid	0.2 g
Omega 3 - EPA	0 mg
Omega 3 - DHA	0 mg
Cholesterol	0 mg
Minerals	**Amount Per Portion**
Calcium	35 mg
Potassium	484 mg
Sodium	6 mg
Copper	405 µg
Iron	2 mg
Magnesium	71 mg
Phosphorus	142 mg
Selenium	1 µg
Zinc	2 mg
Vitamins	**Amount Per Portion**
Vitamin A	37 µg RAE
Vitamin B6	0.2 mg
Vitamin B12	0.0 µg
Vitamin C	14 mg
Vitamin D	0 µg
Vitamin E	4 mg AT
Vitamin K	77 µg
Folate	71 µg DFE
Thiamin	0.1 mg
Riboflavin	0.1 mg
Niacin	2 mg
Choline	21 mg

Serving Tips

- Choose whole wheat pasta.
- Toss with vegetables, such as mushrooms and tomatoes, or chicken.
- Use as a spread on a wrap or sandwich.
- Does not save well as leftovers.

White Bean Chili

Servings: 6

Ingredients

3 chicken breasts (~2 lb.) cut into small cubes

15.2 oz. canned corn, drained

16 oz. canned garbanzo beans, drained

15.5 oz. canned Great Northern beans, drained

32 oz. low sodium chicken broth

1 medium Vidalia onion, diced

~2 Tbsp. minced garlic

~1 Tbsp. olive oil

~1 tsp. ea. oregano, paprika, thyme, and chili powder

Steps

1. In a large skillet or Dutch oven, begin to sauté the onion and garlic with olive oil on medium heat.
2. Add cubed chicken and continue to sauté until the chicken is fully cooked.
3. Drain canned garbanzo beans, Great Northern beans, and corn and add them to the chicken.
4. Add spices and mix well.
5. Add broth and simmer for 20-30 minutes.
6. Serve hot with your choice of toppings.

White Bean Chili

Portions: 6

Food Groups	Amount Per Portion
Grains	0 ounce(s)
Whole Grains	0 ounce(s)
Refined Grains	0 ounce(s)
Vegetables	1 cup(s)
Dark Green	0 cup(s)
Red & Orange	0 cup(s)
Beans & Peas	¾ cup(s)
Starchy	¼ cup(s)
Other	¼ cup(s)
Fruits	0 cup(s)
Fruit Juice	0 cup(s)
Whole Fruit	0 cup(s)
Dairy	0 cup(s)
Milk & Yogurt	0 cup(s)
Cheese	0 cup(s)
Protein Foods	1½ ounce(s)
Seafood	0 ounce(s)
Meat, Poultry & Eggs	1½ ounce(s)
Nuts, Seeds & Soy	0 ounce(s)
Oils	1 teaspoon
Limits	**Amount Per Portion**
Total Calories	354 Calories
Added Sugars	0 Calories
Saturated Fat	11 Calories
Nutrients	**Amount Per Portion**
Protein	29 g
Carbohydrate	46 g
Dietary Fiber	10 g
Total Sugars	5 g
Added Sugars	0 g
Total Fat	7 g
Saturated Fat	1 g
Monounsaturated Fat	3 g
Polyunsaturated Fat	2 g
Linoleic Acid	1 g

α-Linolenic Acid	0.1 g
Omega 3 - EPA	2 mg
Omega 3 - DHA	3 mg
Cholesterol	42 mg
Minerals	**Amount Per Portion**
Calcium	118 mg
Potassium	968 mg
Sodium	657 mg
Copper	476 µg
Iron	5 mg
Magnesium	91 mg
Phosphorus	336 mg
Selenium	18 µg
Zinc	3 mg
Vitamins	**Amount Per Portion**
Vitamin A	21 µg RAE
Vitamin B6	0.6 mg
Vitamin B12	0.2 µg
Vitamin C	4 mg
Vitamin D	0 µg
Vitamin E	2 mg AT
Vitamin K	11 µg
Folate	129 µg DFE
Thiamin	0.2 mg
Riboflavin	0.2 mg
Niacin	7 mg
Choline	96 mg

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Serving Tips

- Choose “low sodium” or “no salt added” canned products and broth.
- Substitute chicken by doubling beans for a vegetarian option.
- Serve with your favorite toppings: tortilla chips, avocado, sour cream, shredded cheese, or bread.
- Make a large batch and save for lunches!

Beef and Broccoli

Servings: 6

Ingredients

1 lb. flank steak or lean sirloin steak cut into strips

¼ cup vegetable oil

~ 2 Tbsp. flour

1 Tbsp. garlic

1 tsp. ginger

2 Tbsp. hoisin sauce

½ cup low sodium soy sauce

¼ cup brown sugar

2 Tbsp. orange juice, calcium added

1 bunch broccoli, chopped

2 cups of rice, cooked

Steps

1. Evenly coat strips of steak with flour, shake off any excess.
2. Heat oil in a large skillet on medium heat. Add steak and cook evenly, about 3 minutes on each side.
3. Remove steak from the pan and reduce heat to low.
4. Add garlic and ginger. Cook until fragrant, about one minute.
5. Add soy sauce, hoisin sauce, brown sugar, and orange juice. Simmer until thick.
6. Add chopped broccoli and cover. Steam broccoli until fully cooked (5-7 minutes).
7. Return steak to the skillet and combine.
8. Serve over rice.

Beef and Broccoli

Portions: 6

Food Groups	Amount Per Portion
Grains	1 ounce(s)
Whole Grains	0 ounce(s)
Refined Grains	1 ounce(s)
Vegetables	1¼ cup(s)
Dark Green	1¼ cup(s)
Red & Orange	0 cup(s)
Beans & Peas	0 cup(s)
Starchy	0 cup(s)
Other	0 cup(s)
Fruits	0 cup(s)
Fruit Juice	0 cup(s)
Whole Fruit	0 cup(s)
Dairy	0 cup(s)
Milk & Yogurt	0 cup(s)
Cheese	0 cup(s)
Protein Foods	2½ ounce(s)
Seafood	0 ounce(s)
Meat, Poultry & Eggs	2½ ounce(s)
Nuts, Seeds & Soy	0 ounce(s)
Oils	2 teaspoon
Limits	**Amount Per Portion**
Total Calories	382 Calories
Added Sugars	40 Calories
Saturated Fat	27 Calories
Nutrients	**Amount Per Portion**
Protein	29 g
Carbohydrate	38 g
Dietary Fiber	3 g
Total Sugars	13 g
Added Sugars	10 g
Total Fat	14 g
Saturated Fat	3 g
Monounsaturated Fat	6 g
Polyunsaturated Fat	4 g
Linoleic Acid	4 g

α-Linolenic Acid	0.6 g
Omega 3 - EPA	2 mg
Omega 3 - DHA	1 mg
Cholesterol	58 mg
Minerals	**Amount Per Portion**
Calcium	93 mg
Potassium	693 mg
Sodium	1211 mg
Copper	163 µg
Iron	4 mg
Magnesium	57 mg
Phosphorus	316 mg
Selenium	29 µg
Zinc	4 mg
Vitamins	**Amount Per Portion**
Vitamin A	33 µg RAE
Vitamin B6	0.6 mg
Vitamin B12	1.3 µg
Vitamin C	93 mg
Vitamin D	0 µg
Vitamin E	2 mg AT
Vitamin K	115 µg
Folate	137 µg DFE
Thiamin	0.2 mg
Riboflavin	0.4 mg
Niacin	7 mg
Choline	80 mg
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Serving Tips

- Serve with brown rice for a serving of whole grains.
- Add additional veggies such as bell peppers, onions, zucchini, or baby corn.
- Garnish with sesame seeds if desired.

Dairy-Free Chocolate Mousse

Ingredients

2 avocados

½ cup canned coconut milk, full fat

¼ cup honey

½ cup unsweetened cocoa powder

2 tsp. vanilla extract

1 tsp. cinnamon

Steps

1. Blend avocados in a food processor until smooth.
2. Add the rest of the ingredients to the food processor.
3. Continue blending until consistent, thick texture is achieved.
4. Pour into cups.
5. Chill for at least 15 minutes before serving.

Dairy-Free Chocolate Mousse

Portions: 4

Food Groups	Amount Per Portion
Grains	0 ounce(s)
Whole Grains	0 ounce(s)
Refined Grains	0 ounce(s)
Vegetables	½ cup(s)
Dark Green	0 cup(s)
Red & Orange	0 cup(s)
Beans & Peas	0 cup(s)
Starchy	0 cup(s)
Other	½ cup(s)
Fruits	0 cup(s)
Fruit Juice	0 cup(s)
Whole Fruit	0 cup(s)
Dairy	0 cup(s)
Milk & Yogurt	0 cup(s)
Cheese	0 cup(s)
Protein Foods	0 ounce(s)
Seafood	0 ounce(s)
Meat, Poultry & Eggs	0 ounce(s)
Nuts, Seeds & Soy	0 ounce(s)
Oils	2 teaspoon
Limits	**Amount Per Portion**
Total Calories	274 Calories
Added Sugars	66 Calories
Saturated Fat	78 Calories
Alcohol	5 Calories
Nutrients	**Amount Per Portion**
Protein	4 g
Carbohydrate	32 g
Dietary Fiber	9 g
Total Sugars	19 g
Added Sugars	17 g
Total Fat	19 g
Saturated Fat	9 g
Monounsaturated Fat	7 g
Polyunsaturated Fat	1 g

Linoleic Acid	1 g
α-Linolenic Acid	0.1 g
Omega 3 - EPA	0 mg
Omega 3 - DHA	0 mg
Cholesterol	0 mg
Minerals	**Amount Per Portion**
Calcium	35 mg
Potassium	589 mg
Sodium	13 mg
Copper	628 µg
Iron	3 mg
Magnesium	86 mg
Phosphorus	146 mg
Selenium	4 µg
Zinc	1 mg
Vitamins	**Amount Per Portion**
Vitamin A	5 µg RAE
Vitamin B6	0.2 mg
Vitamin B12	0.0 µg
Vitamin C	8 mg
Vitamin D	0 µg
Vitamin E	1 mg AT
Vitamin K	15 µg
Folate	64 µg DFE
Thiamin	0.1 mg
Riboflavin	0.1 mg
Niacin	2 mg
Choline	14 mg

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Serving Tips

- Add your own mix-ins and toppings such as: shredded coconut, PB2, berries, nuts, banana, or whip cream.
- Blend before eating dinner so that it is chilled and ready to serve for dessert.
- Does not save well, so only make enough to eat immediately.