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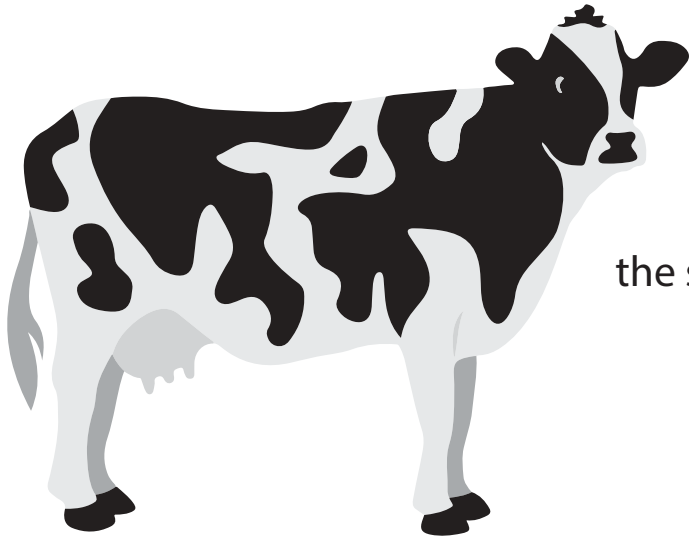
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COW CONUNDRUM

the science behind lactose intolerance

BY Dana Morcillo

We've all heard those words before - lactose intolerant. You, a close friend, or even a random stranger at dhall mutters those words and with it comes the inability to eat milk, cheese, and ice cream. But what does being lactose intolerant actually mean? Besides special access to that one refrigerator in dhall you've always wondered about, lactose intolerance means that you lack a special enzyme needed to digest lactose - a sugar present in many of your favorite foods.

What is lactose and its purpose in the body?

Lactose is a disaccharide, or sugar, made up of glucose and galactose. In order to be broken down into its monosaccharides, it requires a specific enzyme called lactase. Everyone is born with an abundance of lactase in order to break down the lactose present in your mother's milk into needed energy. This lactase resides in the inner lining of your small intestine. It can then break down lactose before it passes into your large intestine. Through this process, glucose and galactose - the resulting sugars - can then enter the small intestine and be absorbed into the bloodstream to be converted or used for energy.

If the body contains little or no lactase, it loses the ability to digest lactose in the small intestine. This results in lactose being passed through the large intestine and into the colon where it's broken down by bacterial fermentation. This process of digestion is called lactose malabsorption. However, lactose malabsorption doesn't always lead to lactose intolerance. That transition can depend on different factors such as how much lactose was consumed, and an individual's colonic flora and sensitivity to lactose fermentation products.

What is lactose intolerance?

Lactose intolerance is a condition in which an individual experiences irregular digestive symptoms such as diarrhea, abdominal pain, or bloating after consuming any food or drink that contains lactose.¹ These painful symptoms are the result of lactose malabsorption and can manifest at three different levels. The most common type is primary lactose intolerance. Primary lactose intolerance is caused by a dropoff in the production of lactase seen in adulthood. This type of intolerance is genetically determined and seen most frequently in individuals of African, Asian or Hispanic ancestry.² Secondary lactose intolerance is a decrease in lactase production caused by damage to the small intestine whether it's through illness, injury or surgery. The last is developmental lactose intolerance. Those with this heritable condition are born without any lactase or lactase activity.

How does one become lactose intolerance?

The majority of us are born with a working lactase gene that produces all of the lactase present in our intestine. Yet, 75% of the world's population suffers from some type of lactose intolerance.³ Why is that? The answer lies in both genetics and epigenetics. The expression of the LCT gene is responsible for the production of lactase. However, as we grow and become older, our diet expands beyond milk to other foods. This transition means that the lactase gene is read and expressed less often. Over time, this leads to a decreased expression of that gene and thus, of lactase in a process called lactose nonpersistence that can lead to lactose intolerance. However, a small portion of the population experiences lactose persistence where they are able to continue producing lactase well into adulthood. This population contains mutations within their LCT gene that is believed to allow for the high lactase activity. However, genetically lactose nonpersistent individuals aren't always lactose intolerant nor are genetically lactose persistent always lactose tolerant.

In a study published in 2016 by Labrie and co-authors, researchers attempted to understand the role epigenetics may play in answering why genetics doesn't lead to the expected expressed phenotypes seen in lactose intolerance. Through profiling and sequencing techniques, the authors looked at and compared the lactase genes in the small intestine of a mouse (which does not experience lactose nonpersistence) and a human. In comparing genes, lactase expression was seen to differ through a hindrance of transcription factors or other components responsible for regulating the gene.⁴ These regulatory sites are epigenetically-controlled suggesting that epigenetic changes that can accompany age contribute to the lack of connection seen between genetics and phenotypic outcome.

How does one treat lactose intolerance?

The first and possibly most difficult solution is to avoid lactose. Despite lactose being present in milk and cheese, new alternatives are available that can allow you to avoid lactose but still enjoy your favorite foods. These alternatives include soy, almond or oat milk, vegan cheese, and even cashew cream cheese. Another popular treatment is through lactase supplements such as Lactaid. Lactaid contains the lactase enzyme and helps break down the lactose present in these lactose-filled foods. The effectiveness of these supplements can be dependent on the person as each individual is different in their sensitivity to lactose and thus, might need a different amount of Lactaid in order for it to be effective. Overall, it's important to remember that every person is unique in their biology so finding a solution that works can be trial-and-error.

SOURCES

¹"Definition & Facts for Lactose Intolerance." National Institute of Diabetes and Digestive and Kidney Diseases, U.S. Department of Health and Human Services, 1 Feb. 2018, <https://www.niddk.nih.gov/health-information/digestive-diseases/lactose-intolerance/definition-facts>.

^{2,3} Scrimshaw, N. S., and E. B. Murray. "The Acceptability of Milk and Milk Products in Populations with a High Prevalence of Lactose Intolerance." *The American Journal of Clinical Nutrition*, U.S. National Library of Medicine, Oct. 1988, <https://www.ncbi.nlm.nih.gov/pubmed/3140651>.

⁴Labrie, Viviane, et al. "Lactase Nonpersistence Is Directed by DNA-Variation-Dependent Epigenetic Aging." *Nature Structural & Molecular Biology*, U.S. National Library of Medicine, June 2016, <https://www.ncbi.nlm.nih.gov/pubmed/27159559#>.



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