helping to repair damaged proteins and enhancing the activity of Chaperones and proteasomes. Sirtuin activation is heavily influenced by cellular NAD+ (nicotinamide adenine dinucleotide) levels. NAD+ acts as a co-substrate for sirtuins, meaning their enzymatic activity depends on its presence. SIRT1 can deacetylate and activate key chaperones like Hsp70, potentially enhancing their protein folding capacity. Poly(ADP-ribose) Polymerase (PARP) Inhibition: During cellular stress, PARPs consume NAD+ to repair DNA damage. Inhibiting PARP activity prevents this NAD+ depletion, making it more available for sirtuins [2,3,4].

Conclusion

Effects of reactive oxygen species (ROS) generated by mitochondria during energy production. We explored the vicious cycle where increased ROS damages the mitochondria itself, leading to further ROS production damaging cellular structure such as sER membrane, and ultimately cell death. The cellular response to this stress includes the activation of chaperones, proteasomes, and sirtuins. Chaperones assist in proper protein folding, while proteasomes degrade damaged proteins. Sirtuins play a central role by influencing both chaperone and proteasome activity, and their activation is dependent on cellular NAD+ levels.

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THE INTRICATE RELATIONSHIP BETWEEN LACTASE ENZYME LEVELS AND LACTOSE INTOLERNCE IN HUMANS

Introduction

Individuals with lactose intolerance don't produce enough lactase, also known as β-galactosidase, necessary for converting milk's lactose into simple sugars, glucose and galactose. Lacking lactase means lactose ferments in the colon, leading to gas and symptoms like bloating, diarrhea, and nausea [1].

Lactase-phlorizin hydrolase (LPH) is crucial for digesting milk's lactose. Post-weaning, most people see a drop in this enzyme, a condition termed lactase non-persistence (LNP), leaving them lactose intolerant. Yet, a minority retain high enzyme levels, a genetically complex trait called lactase persistence (LP), enabling lifelong milk digestion – often in communities with a history of herding [2].

LPH, encoded by the LCT gene on chromosome 2q21, is exclusive to the upper intestine's microvilli and peaks during infancy. Two-thirds of adults worldwide exhibit LNP, while about one-third, particularly those from pastoral cultures, have the LP gene passed down dominantly. LP's genetic underpinnings include several mutations in the MCM6 region near the LCT gene [2].

Moreover, lactose intolerance can lead to calcium deficiency, as the unabsorbed lactose's transformation by bacteria into lactic acid and other compounds could mitigate intolerance effects [2].

Goal

To determine lactose intolerant people and their severity on food intake and genetics of the enzyme lactase.

Material and methods of research

I randomly selected 120 people and collected data through an online self-administered questionnaire using Google Forms. Using scientific literature and articles relevant to the topic.

The result of the research and their discussion

In an examination involving 120 individuals selected by chance, their comprehension of Lactose Intolerance was scrutinized. The study encompassed an analysis of the subjects' social and demographic attributes, including their age, sex, ethnic background, professional status, and awareness concerning Lactose Intolerance. Additional probing inquired about their choices regarding foods with various lactose levels, how often they indulged in such food items, the adverse effects following lactose consumption, the span of discomfort experienced, the onset of initial symptoms, the employment of lactose-free alternatives and products, as well as any familial patterns of Lactose Intolerance in immediate family members along with associated chronic ailments. The participant composition was 66.7% female and 33.3% male.

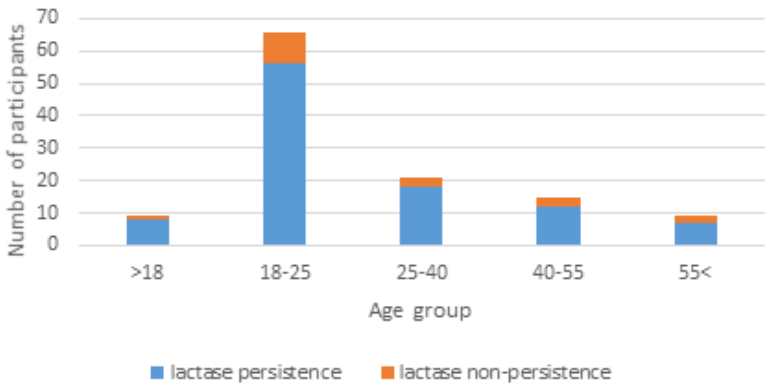

Figure 1 – Comparison of lactose intolerance according to age group

According to the Figure 1 describes the number of participants who are LP and LNP are follows: >18 LP – 8 (88%), LNP – 1 (11.12%); age 18–25 LP – 56 (84.85%), LNP – 10 (15.16%); age 25–40 LP – 18(85.71%), LNP – 30 (14.29%); age 40–55 LP – 12 (80%), LNP – 3 (20%); age 55 < LP7(77.78%), LNP – 2 (22.22%) This indicates that lactose persistence is prevalent across all age groups. And at the age 18-25 has high probability to suffer from lactose intolerance.

As for the country of participants from Sri lanka 80% (n=96). from India 8.34% (n=10), from Belarus 7.5% (n=9), from other countries 4.17% (n=5) among most of Sri Lankans are lactose persistence its indicates that t is hypothesized that natural selection has elicited a prime role in determining the current frequencies of LP in different human communities since the development of cattle domestication

According to figure 2 it shows most frequently reported symptom is diarrhea which is 42.3%, and equal quantities 38.55% shows in bloating and gas and it indicate the level of severity of lactose indolence individuals.

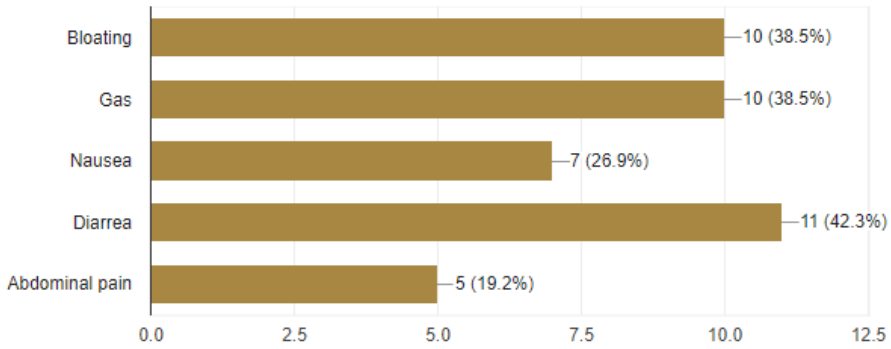

Figure 2 – The symptoms occurrence

When it comes to lactose-rich dairy items, milk (n=103) is the most preferred, followed by your gut (n=97), cheese (n=76), whipped cream (n=21), pudding (n=45), and ice cream (n=87). And the frequency of consumption in the diary shows daily – 55%, 36.7% – sometime, 6.7% – rarely, and 1.7%-not eaten, demonstrating that the majority of people do not have difficulty digesting lactose products and are lactose persistence. However, if a person has LI, the treatment primarily consists of lowering or removing lactose from the diet until the symptoms resolve. As a result, the dietary approach is critical in the management of LI patients, and lactose-free products and supplements are an additional option.83.3% of individuals do not buy lactose-free products, whilst 16.7% do.

Conclusion

The interplay between lactase enzyme production and lactose intolerance is multifaceted and layered. The presence and activity of lactase are crucial for the metabolic processing of lactose, influencing a person's capacity to handle this sugar. Nonetheless, the manifestation of lactose intolerance symptoms can significantly differ among individuals, with contributions from genetic makeup, age, and more. The findings imply that while a predominant number of individuals maintain lactase activity, a minority exhibit a decline, known as lactose non-persistence.

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PATHOLOGY OF THE CARBOHYDRATE METABOLISM: DIABETES MELLITUS AND GLYCOGEN STORAGE DISEASES

Introduction

Carbohydrate metabolism disorders occurs when our body is unable to handle the amount of carbohydrates stored or that is lacking in the body. These disorders impact both the breakdown (catabolism) and synthesis (anabolism) of carbohydrates. Thus, giving rise to these pathologies.