Perspective



Perspective on Fuconomes and Galactonomes

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PERSPECTIVE

Sulfated fucans and sulfated galactans, a relatively recent family of marine fucose- or galactose-framing polysaccharides, must also be recalled for the late glycomics era. These glycans are inherently important since they have strong pharmacological activities in a large number of the structures indicated above. Furthermore, in sea urchins, they are capable of controlling an incredibly rare sugarmediated cell-showing event known as the acrosome reaction. The structure of these glycans is tightly linked to phyla or types of event. Following in the footsteps of the genome, transcriptome, and proteome, the glycome has made strides in research, posing clearly more challenges than the primary undertakings.

Furthermore, in sea urchins, they are capable of controlling an incredibly rare sugar-mediated cell-showing event known as the acrosome reaction. The structure of these glycans is tightly linked to phyla or types of event. Following in the footsteps of the genome, transcriptome, and proteome, the glycome has made strides in research, posing clearly more challenges than the primary undertakings. Galactosemia (from Greek + aemia, meaning galactose + blood, accumulation of galactose in blood) is a rare inherited metabolic condition that inhibits an individual's capacity to correctly metabolise the sugar galactose. Galactosemia is inherited in an autosomal recessive pattern, with a deficit in an enzyme required for proper galactose breakdown. Lactase is an enzyme that breaks down lactose in food into glucose and galactose. Galactosemia occurs when the enzymes required for further galactose processing are significantly reduced or absent, resulting in hazardous quantities of galactose or galactose 1-phosphate in various tissues, as in classic galactosemia, ending in hepatomegaly, cirrhosis, and kidney failure. Infants with galactosemia have a death rate of nearly 75% if they are not treated. Galactosemia is an autosomal recessive condition, which means that a kid must inherit one defective gene from each parent in order to develop the disease. Because they inherit one normal gene and one faulty gene, heterozygotes are carriers. Galactosemia symptoms are absent in carriers. Newborn screening (NBS) for galactosemia is performed routinely in several countries across the world.

This enables a diagnosis to be made when the individual is still a baby. Galactosemia causes lethargy, vomiting, diarrhoea, failure to thrive, and jaundice in infants. None of these symptoms are unique to galactosemia, which frequently causes diagnostic delays. Elimination of lactose and galactose from the diet is the only treatment for classic galactosemia. Despite an early diagnosis and a restricted diet, some galactosemia patients develop longterm consequences such as speech issues, learning problems, neurological impairment (e.g., tremors), and ovarian failure. However, new evidence backs up an idea that Duarte galactosemia can cause language development problems in infants who have no clinical symptoms.

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