# Type IV Galactosemia's Structural and Molecular Biology

**Samantha Banford***

*South Eastern Health and Social Care Trust, Upper Newtonards Road, Dundonald, Belfast, UK*

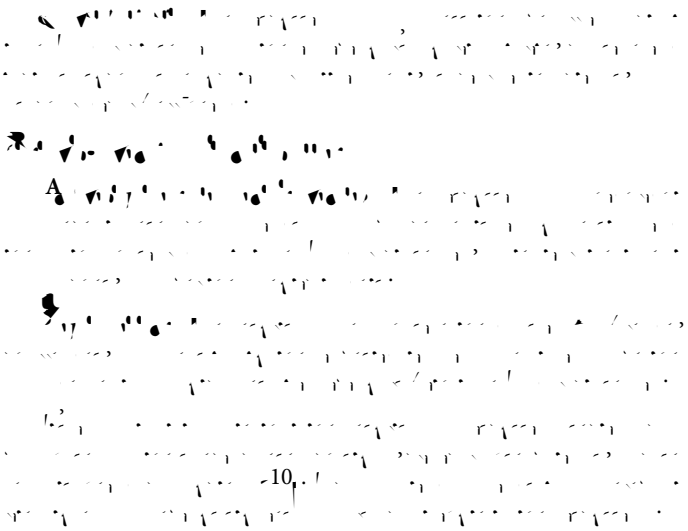
**Abstract**

Type IV galactosemia is a metabolic disorder that is passed down through the family. Galactose autorotate enzyme activity decreases as a result of mutations in the GALM gene. D-galactose and a few other monosaccharides' - and -anomers are interconverted by this enzyme. The structure of human galactose autorotate is largely composed of -sheets and is monomeric. A glutamate acting as a base and a histidine residue acting as an acid are required for the catalytic mechanism. Together, these residues break open the pyranose ring of d-galactose, allowing the monosaccharide's frst two carbon atoms to freely rotate. The hydroxyl group on carbon 1 may reverse its confguration as a result of this. Early onset cataracts are a symptom of type IV galactosemia, which is similar to type II galactosemia (galactokinase defciency). However, as a disease that was only recently discovered, its long-term efects are unknown. It is currently unknown what kind of physiological function, if any, galactose mutarotase's interactions with other monosaccharides play. The potential relationship with different proteins likewise require further examination.

***Corresponding author:** Samantha Banford, South Eastern Health and Social Care Trust, Upper Newtonards Road, Dundonald, Belfast, UK, E-mail: ban.ford@samanath.com

**Received:** 03-June-2023, Manuscript No. jomb-23-104076; **Editor assigned:** 05-June-2023, PreQC No. jomb-23-104076 (PQ); **Reviewed:** 19-June-2023, QC No. jomb-23-104076, **Revised:** 21-June-2023, Manuscript No. jomb-23-104076 (R); **Published:** 28-June-2023, DOI: 10.4172/jomb.1000158

**Citation:** Banford S (2023) Type IV Galactosemia's Structural and Molecular Biology. J Obes Metab 6: 158.




## References

1. Harris AN, Grimm PR, Lee HW, Delpire E, Fang L, et al. (2018) Mechanism of hyperkalemia-induced metabolic acidosis. J Am Soc Nephrol 29: 1411-1425.
2. Palmer BF (2015) Regulation of potassium homeostasis. Clin J Am Soc Nephrol 10: 1050-1060.
3. Szylman P, Better OS, Chaimowitz C, Rosler A (1976) Role of hyperkalemia in the metabolic acidosis of isolated hypoaldosteronism. N Engl J Med 294: 361-365.
4. Mori D, Namiki Y, Sugimachi A, Kado M, Tamai S, et al. (2022) The effect of sodium zirconium cyclosilicate on acid-base balance in chronic kidney disease. Clin Nephrol 97: 255-260.
5. Weir MR, Bakris GL, Bushinsky DA, Mayo MR, Garza D, et al. (2015) Patiromer in patients with kidney disease and hyperkalemia receiving RAAS inhibitors. N Engl J Med 372: 211-221.
6. Velasquez MT, Ramezani A, Raj DS (2015) Urea and protein carbamylation in ESRD: surrogate markers or partners in crime?. Kidney Int 87: 1092-1094.
7. Gorisse L, Pietrement C, Vuiblet V, Schmelzer CEH, Köhler M, et al. (2016) Protein carbamylation is a hallmark of aging. Proc Natl Acad Sci USA 113: 1191-1196.
8. Haldar R, Khandelwal A, Gupta D, Srivastava S, Singh PK, et al. (2016) Acute post-operative diabetic ketoacidosis: Atypical harbinger unmasking latent diabetes mellitus. Indian J Anesthesiol 60: 763-765.
9. Glynn M, Elliot D (1984) Diabetic ketoacidosis and ruptured ectopic pregnancy: a fatal combination. Br Med J 288: 1287-1288.
10. Harris AN, Grimm PR, Lee HW, Delpire E, Fang L, et al. (2018) Mechanism of hyperkalemia-induced metabolic acidosis. J Am Soc Nephrol 29: 1411-1425.