Brief Report
Volume 11:11, 2022

Journal of Metabolic Syndrome

ISSN: 2167-0943 Open Access

Short Note on Chronic Renal Disease

John Fink*

Department of Molecular Biology, University of Kentucky, Lexington, KY 40546, USA

Introduction

kidney diseases are inherited or connected to genetic causes. Due to the lack of distinct clinical symptoms and inadequate therapeutic response, 30 Percent of hospitalised children in the United States with chronic renal failure have hereditary kidney disorders. Hereditary kidney disease has been linked to a number of genes. Hereditary kidney problems must be diagnosed and detected in utero through molecular genetic screening. For more than 30 years, our team has researched inherited kidney disorders. Here is a summary of our group's research on inherited renal illnesses, including our key discoveries. [1].

Description

Many hereditary kidney illnesses have a mendelian inheritance pattern, suggesting that the encoded proteins are necessary for the preservation of renal function. Our understanding of renal development and filtration barrier physiology has considerably benefited from the discovery of genes involved in familial nephrotic syndrome variations and kidney congenital abnormalities. The significant phenotypic and clinical heterogeneity exhibited in monogenic variants of these diseases will be the main focus of this review. We will also emphasise the significance of susceptibility genes in renal disorders with complex inheritance [2].

For a subset of people with hereditary kidney disease, a novel class of aminoglycosides with improved translational readthrough of nonsense mutations and decreased toxicity has been introduced. This approach is ideal for the kidney because aminoglycosides are taken up and maintained there at high intracellular concentrations. In this work, we evaluate the therapeutic window for subclasses of each illness caused by nonsense mutations, as well as the feasibility of aminoglycoside read-through therapy in a variety of hereditary kidney diseases [3].

The various clinical and hereditary patterns of polycystic kidney disease syndromes are brought on by genetic anomalies at various loci. Doctors must use their clinical judgement and experience to determine which patients have these conditions because molecular diagnostics are not yet a practical clinical tool for diagnosing the majority of cystic kidney illnesses. This publication's objective is to inform practising clinicians about the genetic trends, fundamental epidemiology, and phenotypic features of the most prevalent cystic renal diseases. We shall go into detail regarding the following situations: Nephronopthisis-medullary cystic kidney disease complex, Bardet-Biedl syndrome, oral-facial-digital syndrome type 1, autosomal dominant polycystic kidney disease, autosomal recessive polycystic kidney disease [4,5].

Conclusion

About 10 percent of adults and almost all children who require renal replacement therapy have hereditary kidney disease. Both our understanding of renal and syndromic illnesses and our capacity for genetic diagnostics have improved. The genetics of renal diseases, including prevalent monogenic conditions like polycystic kidney disease, Alport syndrome, and Fabry disease as well as more complex conditions including kidney and urinary tract congenital anomalies, are covered in this article. We offer the nephrologist a comprehensive approach to managing inherited diseases, outlining common genetic testing, a manual for genetic counselling, and reproductive alternatives for at-risk couples, such as prenatal or pre-implantation genetic diagnosis.

Acknowledgement

None.

Conflict of Interest

None.

References

1. Phan, T. C. A., Jiake Xu and M. H. Zheng. "Interaction between osteoblast and osteoclast: Impact in bone disease." *Histol Histopathol* 19 (2004).
2. Mayer P., J.L. Pépin, G. Bettega and D. Veale, et al. "Relationship between body mass index, age and upper airway measurements in snorers and sleep apnoea patients." *Eur Respir J* 9 (1996): 1801-1809.
3. Ward, Richard A., Bärbel Schmidt, Jeannine Hullin and Günther F. Hillebrand, et al. "A comparison of on-line hemodiafiltration and high-flux hemodialysis: A prospective clinical study." *J Am Soc Nephrol* 11 (2000): 2344-2350.
4. Semenza, Gregg L., and Reed E. Pyeritz. "Respiratory complications of mucopolysaccharide storage disorders." *Med* 67 (1988): 209-219.
5. Delanaye, Pierre, Bernard E. Dubois, François Jouret and Jean-Marie Krzesinski, et al. "Parathormone and bone-specific alkaline phosphatase for the follow-up of bone turnover in hemodialysis patients: Is it so simple?." *Clin Chim Acta* 417 (2013): 35-38.

How to cite this article: Fink, John. "Short Note on Chronic Renal Disease." J Metabolic Synd 11 (2022): 308.

***Address for Correspondence:** John Fink, Department of Molecular Biology, University of Kentucky, Lexington, KY 40546, USA, E-mail: Johnfink22@gmail.com*

Received: 01 November, 2022, Manuscript No. jms-23-87842; **Editor Assigned:** 03 November, 2022, PreQC No. P-87842; **Reviewed:** 15 November, 2022, QC No. Q-87842; **Revised:** 22 November, 2022, Manuscript No. R-87842; **Published:** 29 November, 2022, DOI: 10.37421/ 2167-0943.2022.11.308