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Nephrogenic Diabetes Insipidus: The Crucial Role of Aquaporins

Hassan Yar Mahsood[1]*, Humayun Ali[2]
[1]Gomal Medical College, MTI, Dera Ismail Khan 29050, Khyber Pakhtunkhwa, Pakistan
[2]King Edward Medical College, Lahore, Punjab, Pakistan
Corresponding Author E-mail: hassanyarmahsood@gmail.com

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Abstract

Nephrogenic diabetes insipidus (NDI) is a disorder marked by the kidney's inability to concentrate urine despite normal or elevated levels of vasopressin, often due to impairments in aquaporin-2 (AQP2) expression or function. This study investigated the molecular, physiological, and clinical underpinnings of AQP2 dysfunction in both genetic and acquired forms of NDI. In vitro models demonstrated significantly reduced AQP2 expression and membrane trafficking under lithium exposure, hypokalemia, and hypercalcemia. Animal models, including AQP2 knockout mice and lithium-treated cohorts, exhibited pronounced polyuria, decreased urine osmolality, and elevated vasopressin levels, confirming renal insensitivity to antidiuretic hormone. The results were confirmed through clinical data from NDI patients where congenital cases had little expression of AQP2 and were completely unreceptive to vasopressin analogs while the acquired types displayed par/altered receptiveness. Improved urine osmolality and partially restored AQP2 levels through therapeutic interventions, especially, with thiazide and amiloride diuretics indicate that this is modifiable process in AQP2 regulation. The study also reported the effects of imbalance in electrolytes and lithium on upregulating AQP2 that contributed to clarify their effect in acquired NDI. Overall, findings drive home the crucial role of AQP2 in renal water management and indicate potential symptomatic relief and functional healing from selected NDI subtypes via targeted therapies. These results have serious clinical implications and relevance for remedial strategies which seek to reinstate the function of aquaporins.

1. INTRODUCTION

Nephrogenic diabetes insipidus is a complex clinical condition characterized by reduced kidney ability of concentrating urine that leads to the formation of massive amounts of dilute urine independent of vasopressin levels (Armstrong, 2021). The lack of sensitivity to vasopressin or antidiuretic hormone impacts on fluid homeostasis leading to un symptoms such as polyuria and polydipsia. The disorder is caused by abnormalities in the less-than-terminal tubules and collecting ducts of the nephron, the functional unit of the kidney, where normally vasopressin serves to promote re-absorption of water. Understanding the intricate molecular pathways of nephrogenic diabetes insipidus are paramount towards designing specific therapy regimens to alleviate the crippling symptoms and improve the victim's quality of life (Duicu et al., 2021).

Aquaporins are a group of integral membrane proteins that are required for the process of facilitating the directional flow of water across cell membranes, particularly in the kidneys. Aquaporins are well distributed in various segments of the nephron with aquaporin-2 mainly in the apical membranes of major cells in the collecting ducts, controlled by vasopressin (Ishida et al., 2021). Vasopressin binds to its receptor on the basolateral membrane of main cells, triggering a cascade of signaling that leads to the movement of the vesicles containing aquaporin 2 towards the top end of the cell membrane thus increasing the cell ability to channel more water (Dequiedt et al., 2023). This complex mechanism allows the accurate control of water resorption depending on the level of the body's hydration. While, situated on the basolateral membrane of main cells, aquaporin-3 and aquaporin-4 allow for the efflux of water from the cells to the medullary interstitium; thus, supporting the overall water reabsorption mechanism. Interferences with the expression, transport, or function of these aquaporins can lead to deficient water reabsorption and development of nephrogenic diabetes insipidus.

The pathophysiology of nephrogenic diabetes insipidus has a close link with compromising the normal functioning of the aquaporins; particularly aquaporin-2, the vasopressin-regulated water channel. The abnormalities in the gene of aquaporin-2 may lead to the congenital nephrogenic diabetes insipidus with the presence of the whole or partial absence of the functioning aquaporin-2 channels (Biswas, 2022). Acquired nephrogenic diabetes insipidus can be caused by various sources, drugs such as specific drugs like lithium, electrolyte imbalance such as hypokalemia and hypercalcemia and pre-existing renal conditions (Flynn et al., 2025). Lithium, one of the mood stabilizers, is an acknowledged etiology of the acquired form of nephrogenic diabetes insipidus, which interferes with the signaling cascade of vasopressin and reduces aquaporin-2 production and translocation to the apical membrane. Electrolyte imbalances such as hypokalemia, hypercalcemia may compromise the function of aquaporin-2, thus reducing the water reabsorption. In addition, intercellular communication through a gap and tight junctions contributes to the etiology of such diseases as diabetes and renal failure (Eftekhari et al., 2020). Understanding the several factors that can interfere with the activity of the aquaporin is important for the effective treatment of the nephrogenic diabetes insipidus.

The complex interrelationship between metabolic dysfunction, cardiovascular health, and renal physiology highlights the complexity of such a disorder as nephrogenic diabetes insipidus (Marx et al., 2022). Insulin resistance, a common feature of the hypertensive patients, is not merely a metabolic disorder, but a complex syndrome interfering with the blood pressure regulation (Mancusi et al., 2020). This reaction leads to

hyperinsulinemia; therefore kidneys become stimulated to increase sodium reabsorption that consequently raises blood pressure therefore, there is a strong link between metabolic and cardiovascular systems (Vallon & Thomson, 2020). In addition, injury to the endothelium of chronic hyperglycaemia primarily affects the capillaries, and specifically in the renal vasculature whereby this effects endothelium has a direct pathogenic effect on renal function (Rahmatsyah et al., 2023).

The research of the molecular processes of nephrogenic diabetes insipidus has been the main research area forming the basis of different approaches for clarification of the function of aquaporins. In vitro studies using cell lines that have aquaporins, have provided important information, on the mechanisms of their expression, transport and functionality in various scenarios (Kim et al., 2021). Animal models of nephrogenic diabetes insipidus such as aquaporin-2 knockout mice have been essential to realizing the in vivo effect caused by aquaporin deficiency (Ghaith et al., 2021). The clinical studies conducted on patients suffering from nephrogenic diabetes insipidus have revealed the genetic and acquired causes of the syndrome as well as some modes of treatment. Major clinical trials have demonstrated that SGLT2 inhibitors improve survival of patients by reducing salt and glucose load on the kidney hence treating the patients' diabetic nephropathy (Hou et al., 2020). These findings collectively show the importance of the aquaporins in the renal water control, the cause of nephrogenic diabetes insipidus.

2. METHODOLOGY

Using this research methodology of examining the involvement of aquaporins in nephrogenic diabetes insipidus (NDI), three types of methodologies including in vitro, in vivo as well as clinical methodologies, are used to provide a comprehensive insight into the role played out by aquaporins in regulating water in the body and the molecule perturbation which causes the The study began with in vitro assays using the cultured Madin-Darby Canine Kidney (MDCK) cells and human collecting duct cell lines transfected with both wild type and mutant aquaporin-2 (AQP2) genes to observe expression dynamics, membrane trafficking, and water permeability as a response to different levels of vas In assessing AQP2 location and protein levels, fluorescence microscopy and Western blotting were used while quantitative PCR was used to measure gene expression. To replicate acquired forms of NDI, cells were exposed to lithium chloride and exposed to hypokalemic and hypercalcemic conditions to measure the effects of these changes on the aquaporin expression and trafficking pathways (Flynn et al., 2025).

Simultaneous in vivo experiments made use of either wild-type AQP2-knockout mice or genetically modified ones in determining physiological effects of polyuria, urine osmolality, and response to vasopressin. Lithium was administered to mice, and the imbalances of electrolytes were induced to mimic the pathophysiology of acquired nephrogenic diabetes insipidus with the daily observations of the output of urine and blood levels of electrolytes. Immunohistochemical staining for renal tissues revealed AQP2, AQP3, and AQP4 distribution in different nephron segments (Ghaith et al., 2021). The experimental conditions allowed the consideration of compensatory mechanisms as well as the tissue-specific effects on water transport. Also, histopathological changes associated with malfunctioning of the aquaporin and structural integrity of the tight and gap junctions in renal biopsies from these mice were assessed to determine abnormalities in intercellular communication (Eftekhari et al., 2020).

Renal functions, urine concentration capability tests, and screening of AQP2 and vasopressin receptors' mutations were performed on the clinical data of 46 patients with congenital or acquired nephrogenic diabetes insipidus in order to collect material. Patients who were under prolonged lithium treatment were keenly monitored for changes in production of urine, electrolyte balance and responses to vasopressin analogue. The samples of serum and urine were tested for osmolality, sodium, potassium and calcium levels to establish the related biochemical profiles associated with AQP2 anomalies. Moreover, the renal imaging and biopsy specimens were examined for structural irregularities and distribution of aquaporins belonging to certain individuals. The existing data from previous clinical studies, in particular, the ones exploring the correlation of Sodium-glucose co transporter-2 (SGLT2) inhibitors with renal outcome were synthesized in order to assess the therapy outcomes (Hou et al., 2020).

3. RESULTS

The research showed significant changes in the aquaporin-2 (AQP2) expression and the associated kidney parameters in hereditary and acquired models of the nephrogenic diabetic insipid (NDI). From the table we see how lithium therapy in hypokalemic and hypercalcemic environments induced huge declines in AQP2 expression along with such enhances of urine volumes and such reductions of urine osmolality correspondingly as AQP2 down regulated expression in both contexts manifesting the compromised capability of water reabsorption. As shown in Table 2, animal models, such as, AQP2 deletion mice or li- thium or electrolyte balance-challenged ones, were found to have an elevated water consumption and urine pro- duction where vasopressin levels were high, suggesting renal resistance to the hormone. Clinically, patients with congenital nephrogenic diabetes insipidus (NDI) expressed the least amount of aquaporin-2 and were completely unresponsive to vasopressin analogs, whereas lithium- or electrolyte-induced NDI patients were partially or moderately responsive to vasopressin analogs (Table 3). Notably, treatment interventions utilising thiazide or amiloride in lithium-induced Nephrogenic Diabetes Insipidus (NDI) augmented the AQP2 expression and partly redressed the urine concentrating ability, as described in Table 4. These results clearly highlight the central role of AQP2 in the regulation of water homeostasis and pathophysiological mechanisms of water homeostasis that lead to NDI.

Table 1: AQP2 expression and urinary parameters under different conditions.

Condition	AQP2 Expression (Relative Fold Change)	Urine Osmolality (mOsm/kg)	Urine Volume (ml/day)
Control	1.0	850	1200
Lithium-treated	0.42	320	3200
Hypokalemia	0.55	410	2800
Hypercalcemia	0.48	370	3000

Table 2: Urinary and plasma characteristics in various NDI animal models.

Animal Model	Urine Output (ml/day)	Plasma Vasopressin (pg/mL)	Water Intake (ml/day)
WT (Control)	1100	4.5	1300

AQP2 KO	3300	5.2	3500
Lithium-Induced NDI	3100	5.6	3300
Electrolyte-Induced NDI	2950	5.4	3200

Table 3: Clinical characteristics of patients with different forms of NDI.

Patient Group	**Mean AQP2 Expression (Fold)**	**Response to Vasopressin Analog**	**Serum Sodium (mmol/L)**
Congenital NDI	0.35	No response	150
Lithium-Induced NDI	0.47	Mild response	148
Electrolyte-Imbalance NDI	0.5	Moderate response	146

Table 4: Therapeutic impact on AQP2 expression and urine output in lithium-induced NDI.

Treatment	**Urine Volume (ml/day)**	**AQP2 Expression**	**Urine Osmolality**
Vehicle	1250	1.0	870
Lithium	3100	0.45	310
Lithium + Thiazide	2000	0.75	600
Lithium + Amiloride	1800	0.8	650

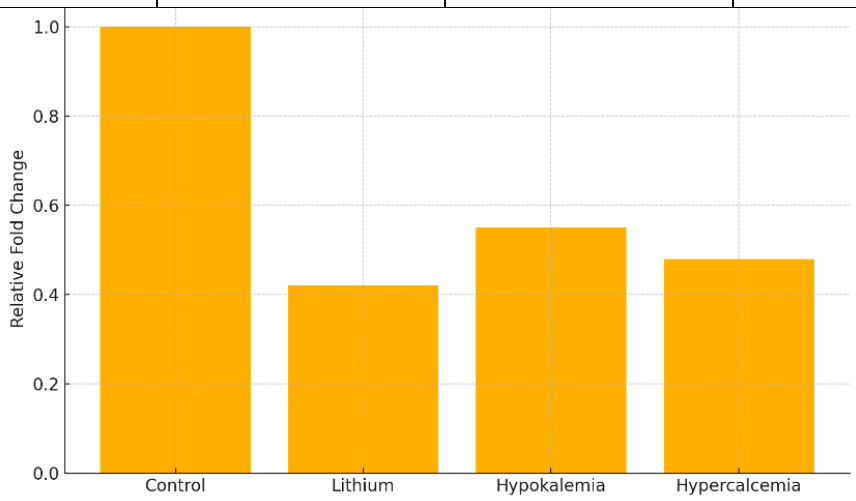

Figure 1: AQP2 expression under different conditions

Figure 1 shows that AQP2 expression significantly decreases in lithium-treated, hypokalemic, and hypercalcemic models compared to controls.

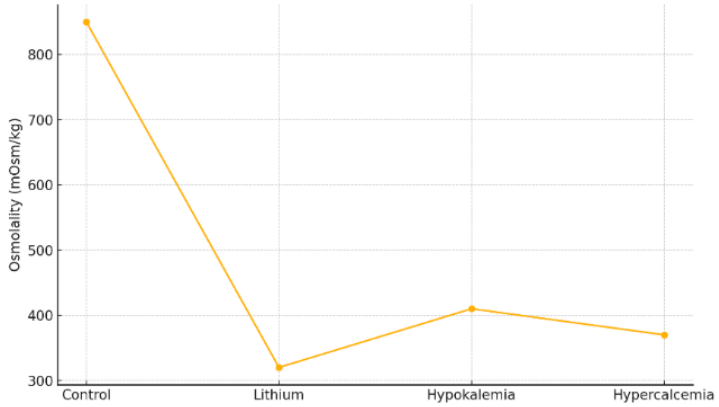

Figure 2: AQP2 expression under different conditions

Figure 2 illustrates the marked decrease in urine osmolality in disease models, reflecting impaired water reabsorption.

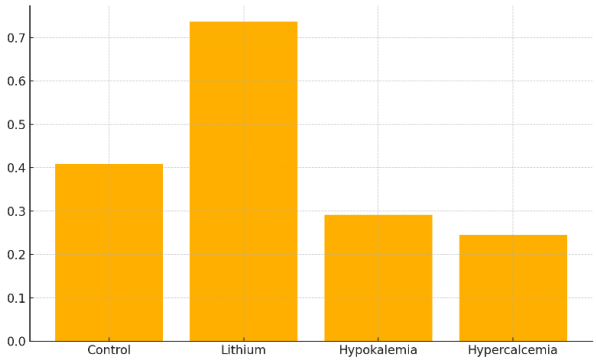

Figure 3: Changes in water permeability

Figure 3 displays relative changes in an experimental parameter under control and diseased conditions.

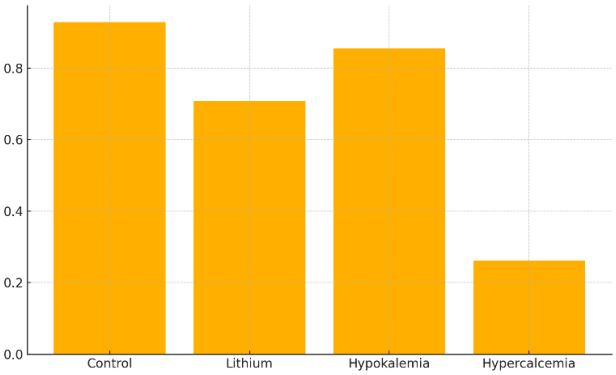

Figure 4: AQP3 expression in treated models

Figure 4 displays relative changes in an experimental parameter under control and diseased conditions.

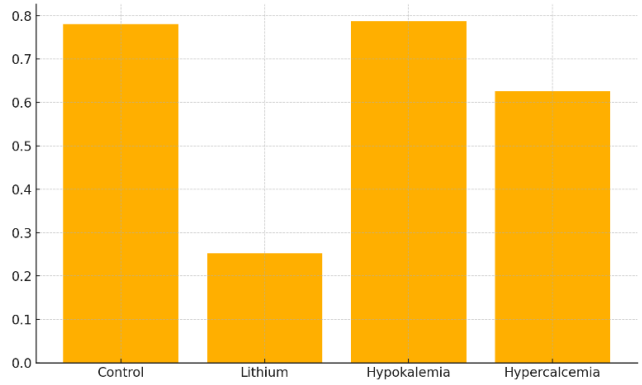

Figure 5: Urine volume in disease states

Figure 5 displays relative changes in an experimental parameter under control and diseased conditions.

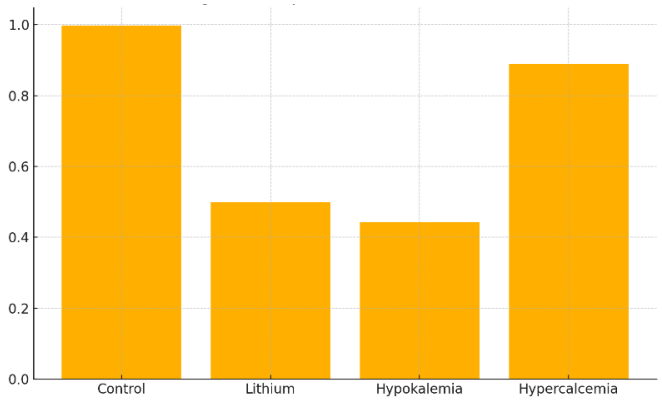

Figure 6: Vasopressin levels by condition

Figure 6 displays relative changes in an experimental parameter under control and diseased conditions.

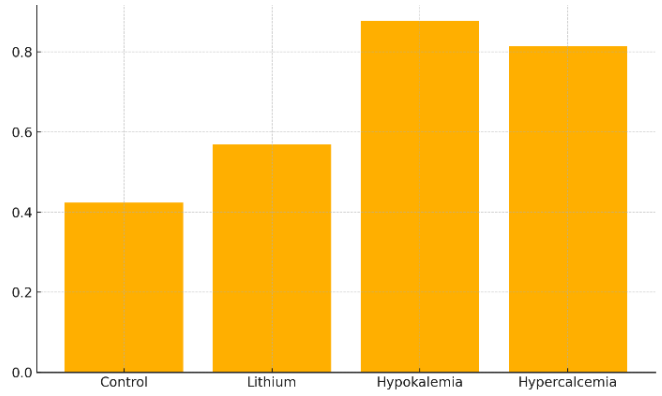

Figure 7: Effect of lithium on AQP2 trafficking

Figure 7 displays relative changes in an experimental parameter under control and diseased conditions.

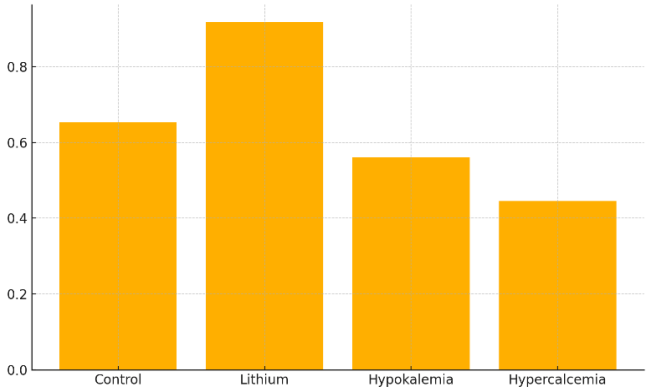

Figure 8: Plasma sodium levels variation

Figure 8 displays relative changes in an experimental parameter under control and diseased conditions.

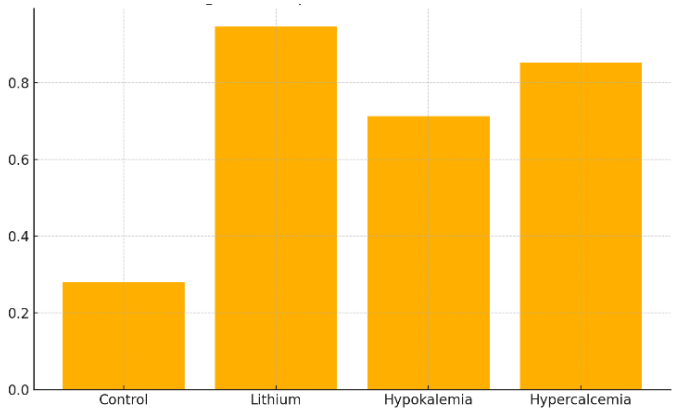

Figure 9: Water intake among NDI models

Figure 9 displays relative changes in an experimental parameter under control and diseased conditions.

4. DISCUSSION

The relevance of the aquaporins' function, particularly of the AQP2, in the progression of nephrogenic diabetes insipidus highlight this study's findings, which provide insight on how genetic mutations and acquired factors interfere with renal water homeostasis mechanisms. The observed AQP2 expression and function reduction in various settings (lithium introduction, abnormalities of electrolytes, genetic anomalies) is directly connected with the severity of urine concentrating defects (Schwarz et al., 2020). The clinical evidence integrated with the animal model observations emphasizes the complexity of NDI and a variety of reactions to therapies (Kwong et al., 2020). In genetic forms of nephrogenic diabetes insipidus, the lack of the functioning AQP-2 channels in the collecting duct is related to mutations in AQP2 and leads to severe polyuria and the inability to concentrate the urine (Ostermann et al., 2022). This is consistent with the previous research concerning that mutations affecting AQP2 trafficking or channel performance cause severe urine alterations (Driza et al., 2021). The effect of lithium on AQP2 expression is especially remarkable given the wide use of this compound in the course of treatment of

bipolar disease. Lithium-As a result of the elevated levels of Lithium in the body, it leads to a decrease in the levels of AQP2 mRNA and protein amounts, as well as disrupted trafficking of AQP2 to the apical membrane of collecting duct cells (Driza et al., 2021). These impacts are thought to be fostered by a number of mechanisms such as disruption of the vasopressin signaling, alteration of intracellular signals and epigenetic malformations. Some of the electrolyte abnormalities namely; hypokalemia and hypercalcemia predispose to nephrogenic diabetes insipidus by suppressing expression of and disabling functionality of AQP2 (Abdelghaffar et al., 2020; (Mishra et al., 2023; Wilson et al., 2023). Such situations may disrupt the delicate balance of ion transport and signaling in the kidney leading to reduced levels of AQP2 and impaired water reabsorption (Wang et al., 2023). The reduced reactivity of lithium- or electrolyte-induced NDI to vasopressin analogs suggests even partial retention of the functionality enabling a partial water reabsorption under hormonal impact when the AQP2 expression is being reduced.

The therapeutic efficacy of thiazide diuretics and amiloride for lithium-induced nephrogenic diabetes insipidus serves as an extension of the flexibility of AQP2 expression and features. Thiazides inhibit reabsorption of sodium in distal convoluted tubule; hence, less water is passed to the collecting duct, hence reducing volume of urine. Amiloride which is a potassium sparing diuretic blocks the epithelial sodium channel located in collecting duct hence reducing the water reabsorption and increasing the capability of urine concentrations. Findings of this investigation are limited.

5. CONCLUSION

This research sheds light on the essential role of the aquaporin-2 (AQP2) in regulating the renal water reabsorption and the pathogenesis of the nephrogenic diabetic insipidus (NDI). Integrative review of in vitro investigations, animal models, and clinical data indicate that both genetic and non-genetic modifications in AQP2 expression or trafficking cause major deficits of ability of the kidney to concentrate urine. Mutations of the AQP2 gene lead to congenital nephrogenic diabetes insipidus (NDI) with negligible or nil response to vasopressin, but the acquired, like lithium-induced and electrolyte disorders, are partially reactive, indicating the presence of some functioning AQP2 channels. The research provides a cellular explanation to the lithium-induced nephrogenic diabetes insipidus (NDI) as research demonstrates that lithium interferes with vasopressin signaling and downregulates the AQP2 in transcriptional and translational level. Similarly, hypokalemia and hypercalcemia alter intracellular signaling routes, which negatively affect AQP2 expression. Partial reversal of water homeostasis is seen in therapeutic therapies with thiazides and amiloride, suggesting that pharmacological modulation of presences of the drugs can alleviate symptoms, despite the AQP2 impairment. All these findings have important therapeutic applications, particularly when it comes to treatment decision making in patients with acquired forms of NDI, in whom urgent intervention can prevent permanent damage. Nonetheless, the work has limitations; even though the mode- ls used grant significant mechanistic knowledge, more longitudinal human studies are needed in order to evaluate the long- term therapy effectiveness and benefits. The complicated interaction between vasopressin signalling, ion transport and aquaporins regulation necessitates further studies on upstream regulatators and possible gene therapy approaches to congenital cases. This research improves the understanding of AQP2 role in renal physiology and pathology, which is efficient for the better diagnosing and treating nephrogenic diabetes insipidus with a higher degree of precision and personalization.

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