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Mini-review

Molecular mechanisms and translational biology of uremic pruritus in chronic kidney disease: A critical analysis of recent advances

Adarsh Kumar Singh[1], Anupma Kaul[2], Manas Ranjan Behera[2]*, Shafaque Asif[1], Ruchika Tondon[3]& Pallavi Prasad[4]

[1]Centre of Biomedical Research (CBMR), [2]Department of Nephrology, [3]Department of Neurology, [4]Department of Pathology, Sanjay Gandhi Postgraduate Institute of Medical Sciences, Lucknow, Uttar Pradesh, India

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Uremic pruritus (UP) is a prevalent and distressing complication in patients with chronic kidney disease (CKD), particularly those undergoing dialysis. Once considered a peripheral symptom of skin dryness or electrolyte imbalance, UP is now recognized as a complex condition involving neuroimmune dysregulation, inflammatory signaling, and altered skin barrier function. Recent experimental studies have identified key molecular mediators such as interleukin-31 (IL-31), tumor necrosis factor-alpha (TNF-α), transient receptor potential channels (TRPV1, TRPA1), and opioid receptor imbalances that contribute to the pathogenesis of pruritus in CKD. Animal models and *in vitro* assays have provided crucial insights into the mechanisms underlying itch generation and neuronal sensitization. This review critically examines recent advances in the molecular biology of UP and explores translational strategies targeting these pathways. Emphasis is placed on experimental findings from the last decade and their relevance in identifying therapeutic targets. Understanding these mechanisms may help guide future development of personalized treatments and improve quality of life for affected patients.

Keywords: CKD-associated pruritus, Renal itch, Pruritogenic cytokines, Neuroimmune modulation, TRP channel signaling, κ-opioid receptor, Skin barrier dysfunction