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A Brief Report on Primitive Cultures, Evolution and Uric Acid

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Introduction

Progressive kidney disease, often known as "maladaptive" renal disease, is characterised by nephron loss, hyperfiltration, and inadequate healing. Evolutionary medicine is a new field that has emerged in the last 20 years and has expanded research horizons. Unlike physiologic (homeostatic) adaptation, evolutionary adaptation is the result of reproductive success and natural selection. Mismatches between phenotype and environment, as well as evolutionary tradeoffs, can both contribute to evolutionary reasons for physically maladaptive responses.

Description

A vulnerable energy-consuming renal tubule and a hypoxic, hyperosmolar microenvironment emerged from evolutionary adaptation to a terrestrial setting. Natural selection supports a successful energy investment strategy: during reproductive years, energy is committed to maintaining nephron integrity, but this decreases with increasing senescence after w40 years of age. Restricted foetal growth or preterm delivery (a life history tradeoff that results in fewer nephrons), evolutionary selection for APOL1 mutations (a tradeoff that provides resistance to trypanosome infection), and modern life experience are all risk factors for chronic kidney disease (Western diet mismatch leading to diabetes and hypertension). Current advancements in genomes, epigenetics, and developmental biology have identified proximal reasons of kidney disease, but slowing renal disease remains a challenge. By addressing the root causes of kidney illness, evolutionary medicine offers a supplementary solution. Evolutionary mechanisms are responsible for significant variations in nephron number at birth, nephron heterogeneity, and changing vulnerability to kidney injury throughout time. New techniques could emerge from combining molecular genetics, evolutionary developmental biology (evo-devo), developmental programming, and life history theory.

Because of the lack of uricase activity caused by multiple mutations in its gene throughout the Miocene era, humans have greater UA levels than other mammals. Furthermore, 90% of the UA filtered by the kidneys is reabsorbed rather than expelled. These findings show that evolution and physiology did not regard UA as a hazardous waste product, but rather as something valuable that must be preserved. This has prompted some researchers to consider the evolutionary benefits of uricase loss and subsequent increase in UA levels. It has been proposed that the evolutionary benefit of UA's high antioxidant action could be longer life expectancy of hominids. According to some writers, the loss of uricase and increase in UA could be a mechanism for maintaining blood pressure during periods of very low salt intake. The oldest hypothesis links a rise in UA to increased intelligence in humans. Finally, UA has been shown to protect against numerous neurodegenerative illnesses, implying that it may have interesting effects on neuronal growth and function. These hypotheses are examined in terms of evolution as well as their clinical significance. UA has some evident negative effects as well as some less visible positive effects as an antioxidant and neuroprotector.

Hypertension is a worldwide epidemic that affects 25% of the population and is a leading cause of stroke, congestive heart failure, and end-stage renal disease. Surprisingly, there is evidence that the increased prevalence of hypertension is a new occurrence in human history, and that it connects with dietary changes linked with Westernization. In this article, we examine the data linking uric acid to the aetiology and epidemiology of hypertension. We specifically examine the evidence that the Miocene uricase mutation that resulted in increased serum uric acid in humans relative to most other animals may have happened as a mechanism to boost blood pressure in early hominoids in response to a low-sodium and low-purine diet.

The global epidemic of chronic kidney disease (CKD) has motivated new techniques to unravel the underlying mechanisms: the incidence of end-stage kidney disease (ESKD) in children is 10 per million, whereas the lifetime risk of ESKD in adults is more than 5%. Diabetic nephropathy is now the most common cause of ESKD in adults, whereas congenital abnormalities of the kidneys and urinary tract (CAKUT) are the most common cause in children. More recently, epidemiologic studies have revealed that incomplete recovery from acute kidney damage (AKI) is a risk factor for developing CKD, and that CKD enhances susceptibility to AKI: the proximal tubule thus becomes a key target of injury and CKD progression. The gradual understanding of kidney anatomy and function, as well as the fundamental role of the nephron in maintaining homeostasis, has led to advances in nephrology. Jean Oliver's elegant morphologic studies in the early twentieth century revealed the development of intermixed hypertrophied and atrophied nephrons in the kidneys of CKD patients, which was a significant finding. Oliver also described the widespread production of atubular glomeruli and aglomerular tubules in the kidneys of patients with severe CKD using microdissection techniques. The emergence of atubular glomeruli as a result of proximal tubular damage is a hallmark of late CKD, according to morphometric investigations of kidneys from patients with CKD due to vascular, glomerular, tubulointerstitial, or toxic etiologies [1-5].

Conclusion

Systems biology could provide fresh evolutionary insights into CKD maladaptation by merging physiologic and genomic approaches across vast sample sizes of numerous species' life histories. New medicines could be developed to target the processes that cause the population of cells in diseased or wounded tissue to be selected. The clues to renal senescence may be discovered in the epigenetic mechanisms of foetal programming, accounting for the transition from paediatric to adult CKD, if kidney research is framed in the perspective of life history theory.

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Conflict of Interest

There are no conflicts of interest by author.

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