COMMENTARY

Adaptive nephrology

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ABSTRACT

Evolutionary medicine, a new field that broadens study boundaries, has evolved in the last 20 years. Progressive kidney disease follows nephron loss, hyperfiltration, and inadequate healing, a process known as "maladaptive." Evolutionary adaptation, in contrast to physiological (homeostatic) adaptation, is the result of reproductive success, which represents natural selection. Environmental mismatch or evolutionary trade-offs can lead to evolutionary reasons for physiologically maladaptive responses. A fragile, energy-hungry renal tubule and a hypoxic, hyperosmolar microenvironment were produced as a result of evolutionary adaption to a terrestrial environment. Natural selection promotes a successful energy investment strategy: through the reproductive years, energy is committed to maintaining nephron integrity, but after age 40, this decreases due to senescence.

INTRODUCTION

The incidence of End-Stage Kidney Disease (ESKD) in children is less than 10 per million, but lifetime risk for ESKD increases to over 5% in adults. Diabetic nephropathy is now the leading cause of ESKD in adults, while Congenital Anomalies of the Kidneys and Urinary Tract (CAKUT) account for the majority of pediatric cases. More recently, epidemiologic studies have shed light on the underlying mechanisms of the growing global epidemic of Chronic Kidney Disease (CKD). The gradual understanding of kidney anatomy and function as well as the crucial function of the nephron in maintaining homeostasis has led to advancements in the field of nephrology. Elegant morphologic research carried out by Jean Oliver in the early 20th century made the significant discovery that CKD (Bright's disease) patients' kidneys develop intermixed hypertrophied and atrophied nephrons. Oliver also described the widespread development of atubular glomeruli and aglomerular tubules in the kidneys of patients with severe CKD using microdissection techniques. Late CKD is characterized by the development of atubular glomeruli as a result of proximal tubular damage, according to later morphometric analyses of kidneys from individuals with CKD caused by vascular, glomerular, tubulointerstitial, or toxic etiologies. Researchers micropuncture investigations showed that after experimental renal ablation, the remaining nephrons go through hyperfiltration, maintaining short-term homeostasis but ultimately leading to glomerulosclerosis, a reaction that is referred to as "maladaptive." As CKD progresses, extensive extracellular matrix deposition in the renal interstitium is recognized as the final common pathway for nephron demise, which is brought on by improperly fixing damaged nephrons. Physiologists anticipate "optimal" homeostatic responses (adaptations) in biological systems; the term "maladaptation" implies departure from an optimal adaptation to environment, a theoretical ideal that more frequently reflects the perspective of the physiologist (or physician). Disease has been viewed throughout the 19th century and to the present day as a disorder of homeostasis, requiring an understanding of its proximate cause, or stimulus (physiologic adaptation). Angiotensin inhibitors, which are frequently employed in decreasing hyperfiltration damage but do not stop progression, were created as a result of this strategy. Angiotensin inhibitors, which are frequently employed in decreasing hyperfiltration damage but do not stop progression, were created as a result of this strategy. Angiotensin inhibitors, which are frequently employed in decreasing hyperfiltration damage but do not stop progression, were created as a result of this strategy. To comprehend how the nephron behaves in CKD, a supplementary paradigm is

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required. This may help researchers find new biomarkers of progression and potent treatments. The ultimate reason of a biologic reaction can be determined by understanding its evolutionary roots through the process of natural selection, in contrast to its proximate cause. The features that improve reproductive success at the expense of disease vulnerability are those that evolutionists search for in ancestral settings and selective forces that determine disease vulnerability. Homer Smith argued in his book from Fish to Philosopher that a series of evolutionary adaptations in our vertebrate ancestors, who transitioned from marine to fresh water environments and ultimately to survival on land, can explain the complex structure of the kidney. The pathophysiology of crystal-related kidney injury. The book, which was written by the foremost American renal physiologist of the middle of the 20th century, discusses how an evolutionary perspective explains how the kidneys' dependence on the daily filtration of 180 L of plasma and their ability to recover 99% of the filtrate. The context of the word "adaptation" has significant implications: physiologic adaptation (adaptive trait) is a homeostatic mechanism that responds to an immediate environmental stimulus, whereas evolutionary adaptation is the result of reproductive success that reflects natural selection. Many of the early advances in renal physiology were based on animal studies, which required an understanding of evolution to apply the results to humans. Under various physiologic situations, quantitative investigation of the link between physiologic and evolutionary adaptation in hemoglobin oxygen saturation in 25 mammals shows that the 2 types of adaptation work on distinct parameters to keep the adaptive ranges in changing contexts. This ground-breaking investigation demonstrates how homeostasis and evolutionary adaptability are related. Although evolutionary theory has historically been used to study comparative anatomy and physiology, an evolutionary approach to disease remained largely ignored until a new field, now known as "evolutionary medicine," was created in the 1990s by G.C. Williams and Randolph Nesse. Williams stated in 1957 that the same genes' other effects must be attributed to natural selection of harmful consequences. He reasoned that antagonistic pleiotropy, in which a gene has a favorable influence during reproductive age but a negative effect thereafter, can explain senescence. He reasoned that antagonistic pleiotropy, in which a gene has a favorable influence during reproductive age but a negative effect thereafter, can explain senescence. Senescence in all species still has its evolutionary roots in antagonistic pleiotropy. Only humans and some whale species have evolved menopause, thus other mechanisms that may be involved in species with prolonged life after reproduction are yet unknown. The process of natural selection of hereditary differences that boost an individual's reproductive success is the central idea of Darwinian evolution, as put forth in The Origin of Species, which was first published in 1859. Over 50% of nephrons arise after ureteral epithelial branching has stopped in the embryonic kidney, which exhibits significant internephron variability. Nephron heterogeneity is associated with changes in vascular and neuronal connections, as well as variations in nephron size and length, and contributes to a number of critical processes of the mature mammalian kidney, including the regulation of sodium, water, phosphate, and osmolar balance. Short loops of nephrons in mammals are highly permeable to urea while long loops are highly permeable to salt, increasing the concentration of urine. The trade-off: Long-loop nephrons have a stronger vascular supply than short-loop nephrons, and the latter are more susceptible to hypoxia and ischemia. An increase in the fraction of sclerotic glomeruli in the general population after reproductive age is correlated with a 50% reduction in the number of nephrons in cadaveric renal transplant donors older than 55 years compared to those younger than 40 years. In healthy kidney transplant donors, hyperfiltration and mild albuminuria are associated with hypertrophied nephrons, whereas hypertension and older age are associated with glomerulosclerosis. Nephron hypertrophy may be caused by either lower congenital nephron endowment or by metabolic risk factors later in life, highlighting the importance of both developmental plasticity and lifestyle environmental factors superimposed on senescence. Globally sclerotic glomeruli appear to atrophy and reabsorb, and the remaining nephrons appear to hypertrophy.