

## Are high protein diets effective on renal function?

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### Abstract:

Protein or amino acid loading causes an increase in renal blood flow and glomerular filtration rate. Hyper filtration in glomerular accelerates the development of chronic kidney disease. For this reason, it is thought that high protein intake may be harmful to the kidneys. Studies on the subject have focused on the effect of protein amount and duration of consumption on renal function. In short-term studies on hypertension, type 2 diabetes and aged people, high protein intake was found to have an impact on glomerular filtration rate and urine albumin excretion and it was determined that this effect depends on the age in healthy people. However, when individuals with pre-hypertension or first stage hypertension were given high protein for six weeks, it was reported that there may be adverse effects on kidney function in long-term due to a significant increase in cystatin. In a long-term study on adult pigs, the glomerular filtration rate was significantly higher in pigs fed with high protein (35.0% of the energy) compared to those fed with normal protein (15.0% of the energy) at the end of the fourth month and proteinuria was observed in pigs in the group fed with high protein. However, at the end of the eighth month, previously observed results were not obtained between the two groups. In animal experiments, glomerular hyper filtration and fusion have been suggested to cause glomerular injury and progressive chronic nephropathy. In another study, creatinine clearance increased by 5-10% at 3 and 12 months in healthy individuals who consumed a high protein diet for two years. The increase in obesity rate has been used for many years with the positive effect of high protein diets on body weight loss. However, besides there are not certain data on the effects of high protein diets, there is no universally accepted definition for high protein intake and long-term human intervention studies are limited. It is believed that there is a need for new studies to address concerns about this issue.

### INTRODUCTION:

Dietary protein intake can modulate renal function and its role in renal disease has spawned an ongoing debate in the literature. At the center of the controversy is the concern that habitual consumption of dietary protein in excess of recommended amounts promotes chronic renal disease through increased

glomerular pressure and hyperfiltration. Media releases often conclude that, "too much protein stresses the kidney". The real question, however, is whether research in healthy individuals supports this notion. In fact, studies suggest that hyperfiltration in response to various physiological stimuli is a normal adaptive mechanism.

The purpose of this paper is to review the available evidence regarding the effects of protein intake on renal function with particular emphasis on renal disease. This review will consider research regarding the role of dietary protein in chronic kidney disease, normal renal function and kidney stone formation and evaluate the collective body of literature to ascertain whether habitual consumption of dietary protein in excess of what is recommended warrants a health concern in terms of the initiation and promotion of renal disease. In the following review, high protein (HP) diets will be defined as a daily consumption of greater than or equal to 1.5 g/kg/day, which is almost twice the current Recommended Dietary Allowance but within the range of current Dietary Reference Intakes (DRIs) for protein. The Institute of Medicine DRI report concluded that there was insufficient scientific evidence for recommendations of an upper limit of protein intake but suggested an acceptable macronutrient distribution range of 10–35% of total energy for protein intake.

While the optimal ratio of macronutrient intake for adults has typically focused on fat and carbohydrate, contemporary discussions include the role of dietary protein. This is particularly true given the recent popularity of high protein diets in weight management. Although the efficacy of these diets with regard to weight loss is still subject to debate, several studies have demonstrated favorable physiological effects. This has led to a substantial increase in protein intake by individuals adhering to contemporary weight loss plans. As a result, the safety of habitually consuming dietary protein in excess of the Recommended Daily Allowance (RDA) has been questioned.

The role of high protein diets in kidney stone formation has received considerable attention. Excessive protein intake increases excretion of potentially lithogenic substances such as calcium and uric acid. Reddy et al. noted that consumption of a high protein diet for six weeks was associated aciduria and urinary calcium and claimed that this constituted increased risk

of stone formation in ten healthy subjects although none of the ten subjects developed renal stones. The severe carbohydrate restriction imposed in this study may have increased keto-acid production thereby contributing acid formation. Since consumption of fruits and vegetables usually produces a marked base load, restriction of these foods subsequent to the diet intervention may have also contributed to the net acid load.

Studies that claim an increased propensity for stone formation as a result of increased protein intake should be taken at face value because propensity is a surrogate marker and does not represent actual stone formation. Further, randomized control trials have not been done to test whether an increased tendency for stone formation is enhanced with consumption of a high protein diet.

Epidemiological studies provide conflicting evidence with regard to the association between protein intake and the predisposition for kidney stone formation. In a prospective study of over 45,000 men, researchers found a direct correlation between animal protein intake and risk of stone formation. However, findings in women are difficult to interpret due to conflicting reports in the literature. While some studies have shown a direct relationship between animal protein intake and risk of stone formation in women, other work suggests an inverse relationship exists.

Conflicting findings regarding the role of dietary protein in kidney stone formation limit the development of universal guidelines with regard to a recommended protein intake for individuals at increased risk for stone formation. It is not likely that diet alone causes kidney stone formation. Rather, metabolic abnormalities are typically the underlying cause. For example, Nguyen et al. found that high intakes of animal protein adversely affected markers of stone formation in those afflicted with a stone causing disorder, while no changes were observed in healthy individuals. It has been suggested that one must have a preexisting metabolic dysfunction before dietary protein can exert an effect relative to stone formation. This notion has been coined the "powderkeg and tinderbox" theory of renal stone disease by Jaeger. This theory asserts that dietary excesses, such as high protein intake, serve as a tinderbox which, only in tandem with a metabolic abnormality (the powderkeg), can bring about stone formation. At the present time, however, evidence showing that a high protein intake is an inherent cause of this renal abnormality or is consistently associated with increased kidney stone formation does not exist.

#### Conclusion:

Although excessive protein intake remains a health concern in individuals with pre-existing renal disease, the literature lacks significant research demonstrating a link between protein intake and the initiation or progression of renal disease in healthy individuals. More importantly, evidence suggests that protein-induced changes in renal function are likely a normal adaptive mechanism well within the functional limits of a healthy kidney. Without question, long-term studies are needed to clarify the scant evidence currently available regarding this relationship. At present, there is not sufficient proof to warrant public health directives aimed at restricting dietary protein intake in healthy adults for the purpose of preserving renal function.