

The Effect of Some Fish Species on Impaired Kidney

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ABSTRACT

This study was designed to know the effect of some fish species on impaired kidney. It was suggested that high-protein diets may have adverse effects on renal function. The specific aimed of this study was to evaluate the effect of some selected protein foods on impaired kidney of albino rats. To achieve this, Twenty four (24) matured male albino rats aged four weeks were obtained from the Animal House, College of Health Sciences, Benue State University (BSU) Makurdi. The selected protein sources, catfish (*Clarias gariapinus*), tilapia fish (*Tilapia niloticus*), crayfish (*Procambarus clarkii*) and clupeid (*Shanya rana*) were purchased and protein samples determined. The (statistical) design of experiments (DOE) was used as an efficient procedure for the experiment and data obtained were analyzed. Results obtained showed that group fed with crayfish had the highest effect of both serum urea and creatinine (14.25 ± 8.09) and (123.62 ± 24.98), then group fed with clupeid and tilapia fish are very much related in its effect with the values (9.27 ± 1.56) and (9.47 ± 1.79) respectively. There was no significant difference observed in the body weight value of all the different groups fed with various protein diets. Mean food intake observed has the highest in the group fed with tilapia fish (22.47 ± 3.20) followed by clupeid (8.05 ± 2.57) and catfish (6.35 ± 1.19). PCV values of all the groups with kidney dysfunction diseases at the end of the experiment except the control group because of the effect of gentamycine induced. In conclusion, the study suggest that catfish could be recommended for patients suffering from early or moderate severe kidney problem since its level of significance is not different from that of the control at <0.05 level of significance.

Keywords: *Effect, Fish species, impaired kidney, Albino rats*

1. INTRODUCTION

Perception in the dietary intake of people with Kidney disease is fundamental to addressing their treatment or for the prevention or progression of disease. Kidney Disease (KD) is a common disorder with increasing prevalence worldwide. Stein K, (2000) stated that potential effects dietary protein consumption on renal function in persons with normal renal function or mild renal insufficiency has important public health implications given the prevalence of high-protein diets and supplementation. Krauss RM, (2000) suggested that a sustained high-protein diets may have adverse effects on renal function but no data support this claim in people with normal renal function or mild renal insufficiency.

However, Hostetter TH, (1981) said there are theoretical reasons for such concern, including the fact that high-protein diet may acutely increase the glomerular filtration rate (GFR), and possibly cause intraglomerular hypertension, which may lead to progressive loss of renal function.

Soroka N, (1998) stated that many clinical studies have been demonstrated, that protein restriction may slow renal function decline compared with usual protein intake in patients with moderate renal insufficiency.

Furthermore, he stressed that these results remain controversial because the largest study of protein restriction in patients with moderate renal insufficiency found no significant benefit, that a recent meta-analysis estimated that among patients with moderate renal insufficiency, GFR decreases by 0.53ml/min less per year in those who follow a low-protein diet compared with those who do not. The authors of this meta-analysis

suggested that the benefits of a low-protein diet might be more apparent with longer follow-up. Some experimental evidence also suggests that animal proteins may play a greater role in the progression of renal disease than vegetable proteins, but not studies have confirmed these results. In experiments in humans, meat proteins acutely increases GFR compared with dairy protein.

2. AIMS AND OBJECTIVES

This study is aimed at evaluating the effect of some selected protein foods on impaired kidney of albino rats. To achieve this aim we set up the following objectives:

- i. The primary purpose of this work is to determine the association between total protein intake and renal function decline over a period of time in albino rats,
- ii. To determine the association between intake of different types of protein and renal function decline,
- iii. To also determine some haematology and biochemical parameters such as packed cell volume (PCV), serum urea and creatinine level of the rats.

3. LITERATURE REVIEW

Kidneys are paired organs of the body with several functions. They are an essential part of the urinary system and also serve homeostatic functions such as regulations of electrolytes, maintenance of acid-base balance, and regulation of blood pressure. They serve the body as a natural filter of the blood, and remove wastes which are diverted to the urinary bladder. The kidneys are an organ that excretes wastes such as urea and ammonium; the kidneys also produce hormones including

calcitriols, rennin, and erythropoietin. Kidneys receive blood from paired renal arteries, and drain into the paired renal veins. Each kidney excretes urine into ureter, itself a paired structure that empties into the urinary bladder.

Kidney Diseases are diverse, but individuals with kidney disease frequently display characteristic clinical features. The Common clinical conditions involving kidney includes; nephritic and nephritic syndromes, renal cysts, acute kidney injury, chronic kidney disease, urinary tract infection, nephrolithiasis, and urinary tract obstruction. Various cancers of the kidney exists; the most common adult renal cancer is renal cell carcinoma. Cancer, cysts and some other renal conditions can be managed with removal of the kidney, or dialysis and kidney transplantation may be options. Although they are not severely harmful, kidney stones can be a painful and a nuisance, Cotran RS, (2005).

3.1 Functions of the Kidney

They kidneys play three major roles:

- i. Removing of waste products from the body, keeping toxins from building up in the bloodstream.
- ii. Producing of hormones that control other body functions, such regulating blood pressure and producing red blood cells.
- iii. Helps in regulating the level of minerals or electrolytes (e.g., sodium, calcium, and potassium) and fluid in the body.

After the blood has circulated through the body, it passes into the kidneys. The kidneys filter waste products and excess salts and water out of the blood, and pass these out of the body as urine. The kidneys also make hormones that control blood pressure, as well as maintain bone metabolism and the production of red blood cells. It's a serious problem when the kidney stops working. Waste products that build up in the body cause imbalances in chemicals needed to keep the body functioning smoothly.

3.2 Excretion of Waste and Acid-Base Homeostasis

The kidneys excrete a variety of waste products produced by metabolism. These include the nitrogenous waste urea, from protein catabolism, uric acid, from nucleic acid metabolism. The two organ systems, kidney and lungs maintain acid-base homeostasis, which is the pH around a relatively stable value. The kidneys give acid-base homeostasis by regulating bicarbonate (HCO_3^-) concentration.

3.3 Kidney Diseases Condition and Causes

There are many different types of kidney diseases. Kidney diseases can lead to end-stage renal disease (ESRD), a condition in which the kidney fails to work normally. People with kidney failure need to receive dialysis or a kidney transplant. The most common causes of kidney disease are diabetes, high blood pressure, and hardening of the arteries and some are cursed by an inflammation of kidneys, called nephritis. This may be

due to an infection or to an autoimmune reaction where the body's immune or defence system attacks and damage the kidneys. Other kidney diseases, such as Polycystic Kidney Disease (PKD) are caused by problems with the shape or sizes of the kidneys (anatomic disorders) while other kidney diseases interfere with the inner workings of the kidneys (metabolic disorders). Most metabolic kidney disorders are rare, since they need to be inherited from both parents. Other common causes of kidney failure include certain medications that can be toxic to kidney tissue, and blockages of the system that drains the kidneys (which can occur with prostate problems).

3.4 Symptoms and Complications of Kidney Disease

The kidney disease symptoms depend on the type of disease that a person has. If the person develops a high fever is caused by bacterial infection. Some signs of kidney disease include passing too much or too little urine, or passing blood or abnormal chemicals in the urine. When the toxic accumulate in a person's blood, symptoms may include: puffy eyes, hands and feet (called edema), High blood pressure, Fatigue, Shortness of breath, Loss of appetite, Nausea and vomiting, Thirst. Kidney disease does not usually cause pain, but in some cases pain may occur. A kidney stone in the ureter (a tube leading from the kidney to the bladder) can cause severe cramping pain that spread from the lower back to the groin. The pain disappears once the stone has moved through the ureter. This condition occurs when the kidney is working at less than 10% of full capacity. At this stage, the person will need dialysis or kidney transplant to be able to go on living.

3.5 Kidney Failure

Generally, humans can live normally with just one kidney, as one has more functioning renal tissue than is needed to survive. Only when the amount of functioning kidney tissue is greatly diminished will chronic kidney disease developed. Renal replacement therapy, in the form of dialysis or kidney transplantation, indicated when the glomerular filtration rate has fallen very low or if the renal dysfunction leads to severe symptoms.

3.6 Assessment of Renal Function

Patients with kidney disease may have a variety of different clinical presentations. Some have symptoms that are directly referable to the kidney (gross hematuria, flank pain) or to extra renal symptoms (edema, hypertension, signs uremia). Many patients, however, are asymptomatic and noted on routine examination to an elevated serum creatinine concentration or an abnormal urinalysis. This topic will provide an overview of the issues concerning assessment of the GFR in the patient with chronic kidney disease. The utility of the urinalysis, radiologic studies, and kidney biopsy are discussed separately, as is the general approach to the patient with kidney disease.

3.7 Proteins

Proteins are a group of highly complex organic compounds found in all cells. Proteins are made of large molecules containing smaller chains of peptides, which chains of amino acids that are biochemically bonded together. The nutritional value of protein is measured by how well it meets the nutritional needs of the body (Halley L, 1997).

High quality proteins is usually of animal origin, easily digested and contain all essential amino acids in the proportions required by the body. Proteins are crucial to a variety of body functions and structure some of which include growth and repair of body cells tissues, synthesis of enzymes, plasma protein, antibodies (immunoglobulin) and some hormones, and provision of energy, usually a secondary function (Halley L, 1997).

3.8 Effect of High-Protein Diets on Impaired Kidney

Dietary protein intake can modulate renal function and its role in renal disease has spawned an ongoing debate in the literature. At centre of the controversy is the concern that habitual consumption dietary proteins in excess of the recommended amounts promote chronic renal disease through increased glomerular pressure and hyper filtration. Media releases often concludes 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 hyper filtration in response to various physiological stimuli is a normal adaptive mechanism (King AJ, 1993).

The purpose of this paper is to review the available evidence regarding the effects of proteins intake on renal function with particular emphasis on renal disease. This review will consider research regarding the role 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 in excess of what is recommended warrants a health 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 1.5g/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 included that there was insufficient scientific evidence for an upper an upper limit of proteins 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 (Layman DK, 2003), contemporary discussions include the role of dietary protein; this particularly true given the recent popularity of high protein diets in weight management (Fine EJ, 2004). Although the efficacy of these diets with regards 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 (Layman DK, 2003).

3.9 Protein Sources

Four different types of protein sources were selected for this study. They are as follows:

3.9.1 CATFISH (*Clarias Gariapiinus*):

Catfish is eaten in a variety of way; in Europe it is often cooked in similar ways to carp, but in the United States it is typically crumbed with cornmeal and fried (Jenny, 1988). Catfish is eaten in a variety of way; in Europe it is often cooked in similar ways to carp, but in the United States it is typically crumbed with cornmeal and fried (Jenny, 1988). Catfish have been widely caught and farmed for food for hundreds of years in Africa, Asia, Europe, and North America. Judgments as to the quality and flavor vary, with some food critics considering catfish as being excellent food, others dismiss them as watery and lacking in flavor (Jenny 1988).

3.9.2 CLUPEID (*Shanya Rana*):

Clupeids are mostly marine forage fish, although a few species are found in freshwater. No species have scale on the head, and some are entirely scale less. The lateral line is short or absent, and the teeth are usually small where they are present at all. Clupeids typically feed on plankton, and range from 2cm (0.79Inch) to 75cm (30Inch) in length (Froese, 2008). Clupeids spawn huge numbers of eggs (up to 200,000 in some species) near the surface of the waters. Changes in protein, ash and moisture percentages within an age-class of a species were primarily the result of changes in fat percentage (Nelson 1998).

4. MATERIALS AND METHODS

4.1 Materials

The aim of this study is to evaluate the effect of some selected protein foods on impaired kidney of albino rats and the following materials are used for the study:- Spectrophotometer (Jenway, 5061 model), Bench centrifuge (Shanghai surgical factory), Analytical weighing balance (Scout pro spv 407, pine brook USA), Desiccators, Microhaematocrit centrifuge, Capillary Microhaematocrit tube reader, Steam water bath, Test tube/rack, Centrifuge tube, Mortar and pestle, Insulin syringe and needles (1ml), Surgical blades, Beakers, Measuring cylinder, Pasteur

4.2 Chemicals and Reagents

The chemical and reagents used for the research are Gentamycin (80mg), Methylated spirit, Chloroform, Radox diagnostic kit for creatinine and urea test, Distilled water.

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4.3 Collection and Preparation of Experimental Diets

The following protein sources was collected and prepared as experimental diets for the research, catfish (*Clarias gariepinus*), tilapia fish (*Tilapia nitlocus*), crayfish (*Procambarus clarkii*) and clupeid (*Shanya rana*) were purchased from the Jimeta Modern Market in Adamawa State. The protein samples were dried and crushed using a mortar and pestle.

4.4 Experimental Animals

Twenty four (24) matured male albino rats aged four weeks were obtained from the Animal House, College of Health Sciences, Benue State University (BSU) Makurdi, Benue State. The rats were transferred to the Biochemical Laboratory of the Department of

Biochemistry, Federal University of Technology Yola, Adamawa State. The rats were housed under standard laboratory condition where they were allowed to acclimatize for one week until the commencement of the experiment. Normal laboratory rat feed and tap water ad libitum were provided.

5. METHODS

The rats were randomly distributed into six (6) groups A, B, C, D, E and F, four rats in each group with group A serving as control and group B as experimental control as shown in the experimental design below,

5.1 Experimental Design

VARIABLES

Group A control
Group B experimental control
Group C induced rat
Group D induced rat
Group E induced rat
Group F induced rat

TREATMENT

Normal feed
Normal feed
Normal feed & dried clupeid (20%)
Normal feed & dried tilapia (20%)
Normal feed & dried crayfish (20%)
Normal feed & dried catfish (20%)

Induction of kidney damage:- Gentamycine (0.20± 0.02ml/kg/body weight) were administered to groups B, C, D, E and F (aged 7 weeks and weighing from 70g to 120g by subcutaneous injection for five consecutive days).

Diet treatment:- On the seventh day post-induction, groups B, C, D, E and F (induced rats) were fed 50g of normal rat feed incorporated with high protein diet of the experimental diets (20%). Group C was fed with dried clupeid, group D dried tilapia fish, group E dried crayfish and group F dried catfish respectively. The control group A and experimental control B were assigned some grams of the normal rat feed and the feeding continued for a period of weeks.

SUB-CHRONIC STUDIES:- The packed cell volume (PCV) of the animal was determined using blood samples collected via the tail before diet allocation. On the second week of feeding, trial PCV was also determined. At the end of the fourth week, the animals were weighed and then anaesthetized. They were bled with cardiac puncture and the blood sample collected into specimen bottles. Part of the whole blood sample were use for PCV

determination while the remaining blood sample were centrifuge and serum separated using Pasteur pipette into clean and labeled sample bottles for serum creatinine and urea determination.

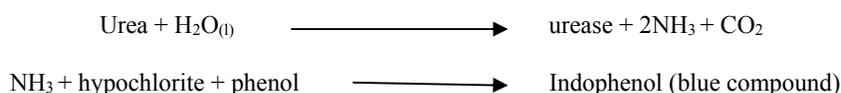
PROCEDURE:- The heparinised capillary tubes were filled with blood samples (2/3 filled) from a capillary puncture. One end of the capillary were sealed with modelling clay (plasticine) and were then placed in the Microhaematocrit centrifuge and spun at 4000rpm for 15 minutes. The spun tubes were placed on the

Microhaematocrit reader and PCV determined in percentage (%).

5.2 Determination of Serum Urea Concentration by Urease (Berthelot Method)

Urea is synthesized in the liver from carbon dioxide and ammonia resulting from the metabolism of amino acid. Urea is carried by the plasma to the kidney where it is filtered and then excreted.

THE TEST PRINCIPLE:- Urea in serum is hydrolyzed to ammonia in the presence of urease. The ammonia is then measured photo metrically by Berthelot's reaction



SAMPLE MATERIAL: SERUM

PROCEDURE:

Wavelength 546nm (530 – 510nm)
Cuvette 1cm length path

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Temperature
Measurement against reagent blank
Pipette into test tubes,

37°C

	Blank	Standard	Sample
Sample	-	-	10µl
Standard	-	10µl	-
Distilled water	10µl	-	-
Reagent 1	100µl	100µl	100µl

Mix and incubate at 37°C for 10 minutes

B	Blank	Standard	Sample
Reagent 2	2.50ml	2.50ml	2.50ml
Reagent 3	2.50ml	2.50ml	2.50ml

Read absorbance of the sample (A sample) and standard (A standard) against the blank.

complex formed is directly proportional to the creatinine concentration.

CALCULATION:- Urea concentration = A sample/A standard × standard concentration (mg/dl)

PRINCIPLE:- Creatinine in alkaline solution reacts with picric acid to form a coloured complex. The amount of the

SAMPLE MATERIAL: SERUM

Wavelength 492nm (490nm – 510nm)
Cuvette 1cm light path
Temperature 25°C/30°C/37°C
Measurement against air

	Standard (semi micro)	Sample (semi micro)
Working reagent	1.0µl	1.0µl
Standard solution	0.1µl	-
Sample	-	0.1µl

Mix and after 30 seconds read the absorbance A1 of the standard and sample. Exactly 2 minutes later, read absorbance A2 of standard and sample.

various groups were subjected to student's test at 5% level of significance.

CALCULATION:- $A_2 - A_1 =$ change in A sample OR change in A standard

Concentration of creatinine = change in $A_{\text{sample}} /$ change in $A_{\text{standard}} \times$ standard concentration (mg/dl).

7. RESULTS

The results obtained from the experiment are summarized below. Table 1 shows the result of the mean body weight for the period of the experiment according to their different diet groups. Increases in the mean body weight were observed in all the different groups of rat. However, the body weight gained was not significant among the rats. In groups B, C, D, E and F, the greatest mean body weight after first and second at 5% level of significance.

6. STATISTICAL ANALYSIS

The result obtained from the experiment will be represented as mean ± SEM of the number of sample. Comparison of the level of food intake, body weight, hematological and biochemical indices of the rats in the

Table 1: Mean Body Weight and Body Weight Gain in Grams

Group	initial body weight	Final body weight	Body weight gain %	Body gain
A = Control	93.00 ± 7.98	107.48 ± 10.26	14.48	15.57
B = Expt.Control	97.10 ± 6.35	120.36 ± 11.58	23.26	23.95
C = Treatment	87.42 ± 7.55	102.03 ± 12.20	20.61	23.58
D = Treatment	86.10 ± 8.18	105.70 ± 9.25	19.60	22.76
E = Treatment	98.45 ± 9.40	112.93 ± 13.87	14.48	14.71
F = Treatment	84.45 ± 11.70	108.90 ± 15.52	24.45	28.95

A = Normal feed

C = Normal feed and clupeid (20%)

E = Normal feed and crayfish (20%)

B = Experimental control and normal feed

D = Normal feed and tilapia (20%)

F = Normal feed and catfish (20%)

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Table 2 shows the mean food intake of the rats according to the different diet groups. At the beginning of the experiment, the highest mean food intake of the rats with kidney dysfunction was observed in group E (28.20 ± 4.18) which was significantly higher than the mean food intake of the rats on diets C, D and E and that of normal control, group A.

However, there was a decrease in the mean of food intake of each group with group E having the highest mean food intake (22.47 ± 3.20) followed by the rats on diet C (8.05 ± 2.57) and group F (6.35 ± 1.19). A comparison of the mean food intake of the rats showed that they were significant difference at the 5% level of significance ($P < 0.05$).

Table 2: Mean Feed Intake in Grams

Group	Initial (before damage)	Final (after damage)
A = control	14.47 ± 2.91	10.10 ± 1.97
B = Experimental. Control	13.52 ± 3.45	14.87 ± 2.97
C = Treatment	12.47 ± 2.32	8.05 ± 2.57
D = Treatment	8.52 ± 1.20	5.45 ± 3.93
E = Treatment	28.20 ± 4.18	22.47 ± 3.20
F = Treatment	10.15 ± 1.09	6.35 ± 1.19

A = Normal feed

C = Normal feed and clupeid (20%)

E = Normal feed and crayfish (20%)

B = Normal feed

D = Normal feed and tilapia (20%)

F = Normal feed and catfish (20%)

Table 3 shows the base (0) start and end of weeks of research mean PCV of the rats according to the different diet group. After a week of diet treatment, the highest mean PCV in the rats with kidney dysfunction was observed in rats on diet F ($51.00 \pm 1.29\%$) followed by the rats on diet B ($50.73 \pm 2.45\%$) in descending order which were significantly different than the mean PCV of the normal control group.

However, at the end of the experiment, there was a decrease in mean PCV of the rats on the various diet groups when compared to the mean PCV obtained in the first week of experiment. The highest mean PCV at the end of the feeding was observed in the rats on diet C ($47.00 \pm 1.71\%$), while the least mean PCV was observed in rats on diet F ($43.50 \pm 1.41\%$). Comparison of the individual diets on PCV was significantly different ($P < 0.05$).

Table 3: Mean Packed Cell Volume (Pcv %)

Group	Base line	After 2 weeks	After 4 weeks
A = control	45.30 ± 2.98	46.00 ± 1.29	48.10 ± 3.30
B = Experimental. Control	42.10 ± 2.73	46.50 ± 1.20	45.00 ± 1.22
C = Treatment	45.50 ± 1.40	50.75 ± 2.45	47.00 ± 1.71
D = Treatment	45.30 ± 1.81	47.50 ± 0.96	44.25 ± 2.89
E = Treatment	47.00 ± 2.70	49.00 ± 1.73	46.50 ± 1.15
F = Treatment	43.30 ± 0.29	51.00 ± 1.29	43.50 ± 1.41

A = Normal feed

C = Normal feed and clupeid

E = Normal feed and crayfish

B = Normal feed

D = Normal feed and tilapia

F = Normal feed and catfish

Table 4 shows the mean serum urea and creatinine levels of the rats according to the different diet groups. The rats with kidney dysfunction on diet F ($7.55 \pm 0.71\text{mg/dl}$) had the least serum urea while the one on group E ($14.25 \pm 8.09\text{mg/dl}$) and D ($9.47 \pm 1.79\text{mg/dl}$), which were not significantly different. But the F can be compared to the normal control because the mean is not significantly different from A.

Similarly, serum creatinine level was markedly elevated in the rats on diet E ($123.62 \pm 24.98\text{mg/dl}$) while rats on diet D ($92.82 \pm 13.60\text{mg/dl}$) had the least serum creatinine level followed by the rats on diet C ($112.75 \pm 8.48\text{mg/dl}$) and group F ($101.62 \pm 11.48\text{mg/dl}$) which were higher than the normal control group A. Comparing the effect of the individual diet on the serum creatinine level of the rats were significantly different ($P < 0.05$).

Table 4: Serum Urea and Creatinine Concentration

Group	Urea (mg/dl)	Creatinine (mg/dl)
A = control	5.00 ± 1.35	79.60 ± 14.37
B = Experimental. Control	6.45 ± 1.00	86.56 ± 10.20
C = Treatment	9.27 ± 1.56	112.75 ± 8.48*
D = Treatment	9.47 ± 1.79	92.82 ± 13.60
E = Treatment	14.25 ± 8.09*	123.62 ± 24.98*
F = Treatment	7.55 ± 0.71	101.62 ± 11.45

All values are represented as mean ± SD * significantly higher compared to control

A = Normal feed

C = Normal feed and clupeid

E = Normal feed and crayfish

B = Normal feed

D = Normal feed and tilapia

F = Normal feed and catfish

8. DISCUSSION

The findings from this study indicate that in renal damage, protein restriction slows the loss of renal function while a high protein diet accelerates the loss of renal function. It is generally known that not all protein food sources are of equal value to the body and that both the quality of the dietary proteins is important in the development and progression of chronic renal failure (CRF). At present the most reliable method of estimating changes in renal function is to monitor the rate of rise of creatinine concentration. Increase in mean body weight was observed in all the different group of rats, but the pattern of body weight gain among the rats varied. Table 1 shows the mean body weight of the rats. Significant increase was observed in the group fed with crayfish (112.93 ± 13.87g) followed by the group fed with catfish (108.90 ± 18.52) respectively. Similarly, the least mean body weight was observed in the group fed with clupeid (102.03 ± 12.20g) followed by the group with tilapia (105.70 ± 9.25g) respectively.

More so, Table 2 shows the mean food intake of the rats in which the highest mean food intake was observed in the group fed with crayfish (22.47 ± 3.20g) which correlate with the result obtained in the body weight, closely followed by the group fed with clupeid (8.05 ± 2.57g) and the group fed with catfish (6.35 ± 1.19g) respectively. This was as a result of the effect of the various foods on the impaired kidney of the rats or the amino acid profile of the food. Table 3 revealed the mean PCV of the rats. A trend of increase was observed in the various groups at the start of feeding with the group fed with clupeid (51.00 ± 1.29%) having the highest value followed closely by the group fed with catfish (51.00 ± 1.29%) respectively. However, at the end of the experiment, there was a decrease in the PCV of each group fed with clupeid (47.00 ± 1.71%) having the highest value and crayfish (46.50 ± 1.15%), which were not significantly different at the 5% level of significance (P<0.05). This decrease was as a result of increase in age of the rats.

Table 4 shows the mean serum urea and creatinine level of the rats. The highest serum urea levels

were observed in the group fed with crayfish (14.25 ± 8.09mg/dl) followed by groups fed with tilapia (9.47 ± 1.79mg/dl) and clupeid (9.27 ± 1.56mg/dl), while the least serum urea level was observed in the group fed with catfish (7.55 ± 0.71mg/dl). Similarly, the highest serum creatinine levels were observed in the group fed with catfish (123.62 ± 24.98mg/dl) followed by group fed with clupeid (112.75 ± 8.47mg/dl), while the least value of serum creatinine were observed in the group fed with tilapia followed by the group fed with catfish (101.62 ± 11.45mg/dl) respectively. The high values of serum creatinine obtained in the group fed with crayfish and clupeid indicates under excretion as a result of partially damage kidneys of the rats, which shows that crayfish and clupeid cannot be tolerated by patients with kidney problem.

In order to maintain adequate supply of essential amino acids, Giovenetti (1985) and Wilkins (1996) recommended about 60 – 75% of the protein needs from protein of high biological value, which should be in form of meat, fish and poultry for patients with CRF or other renal diseases.

Varied level of increase in mean body weight, reduction in food intake was noted in all the rat groups compared to their initial values which indicate that there is a significant difference in the body weight. The group on crayfish diet ate more food and had better body weight than the rats fed with other diet. Reduction in food intake and low body weight gain was apparent in rats fed with catfish.

It seems that from this findings, rats with substantial reduction in kidney function, even when placed on low protein diet will definitely present renal responses such as proteinuria, serum urea and creatinine elevation which indicates progressive deterioration in kidney function. However, the severity of this renal response could influence by the protein food source in the diet ingested. For instance, the rats fed with catfish and tilapia had improved renal function than those crayfish and clupeid. Thus, it is clear from this study that the progression of renal damage is certain, but the rate of deterioration could be attenuated when a low protein diet

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is adopted. Also, the degree of uremic symptoms amelioration could be related to the specific protein in the diet. Finally, this study suggest that the choice of protein foods could be a major factor in the rate at which symptoms of chronic renal failure are reduced with low protein diets.

9. CONCLUSION AND RECOMMENDATIONS

Protein restricted diet slows the progress of chronic renal failure while high protein diet accelerates the progress of renal insufficiency, also dietary protein restriction serves as therapy for the prevention or slowing down of renal insufficiency should be encouraged especially in developing countries.

Base on the result obtained from the study, catfish could be recommended more followed by tilapia in the diets of patients with chronic renal insufficiency since the severity of the biochemical parameters (serum urea and creatinine levels) of the rats with renal insufficiency was reduced by the types and biological quality of proteins.

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