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The effects of a controlled Food Borne mediated exposure to Cyanide and Cadmium on antioxidant enzymes and some renal indices in Rats*Kadiri, Helen Ejiro¹ and Ekayoda, Ovigueroye¹**Department of Biochemistry, Faculty of Science, Delta State University, Abraka, Delta State¹ Author for Correspondence: hekad@yahoo.com/ +234-806-046-5365***Abstract**

The study was carried out to investigate the effects of a controlled Food Borne mediated exposure to Cyanide and Cadmium on renal indices in Rats. Twenty healthy Wistar strain male albino rats were divided into four groups with five rats in each group. Group 1 (control) – Rats were given control compounded diet. Group 2 (cyanide) – Rats were given cyanide-contaminated compounded diet. Group 3 (cadmium) – Rats were given cadmium-contaminated compounded diet. Group 4 (cyanide + cadmium) – Rats were given cyanide + cadmium-contaminated compounded diet. The experiment was carried for 28 days and water was given *ad-libitum* throughout the experimental period. After the experiment, the animals were sacrificed and blood samples taken for analysis for creatinine, lipid peroxidation (LPO) GPx, GSH, GR, superoxide dismutase (SOD) and catalase activity. The result obtained showed an antagonistic increase in creatinine, lipid peroxidation (LPO) GPx, GSH and GR levels in the kidney of rats exposed to both toxicants. A significant decrease was also observed in the superoxide dismutase (SOD) and catalase activity in Groups D rats however this decrease was antagonistic when compared to Groups B and C given Cyanide and cadmium only respectively. Thus, the findings of the study indicate that exposure of a mixture of cadmium and cyanide via the food chain causes damage to the kidney of the rats. However, their combined effect was antagonistic.

Keywords: *liver; cyanide; cadmium; oxidative stress; toxicity.*

Introduction

Food chain aids the absorption and distribution of toxicants in humans and this poses a threat which has received attention in recent times. The introduction of these toxicants into the food-chain may bring about toxicity hence studies concerning its bioaccumulation through the food chain have been given much attention (Ezedom and Asagba 2016; Okafor *et al.*, 2002).

Cyanide is a chemical compound which is highly toxic and it is present in some industrial effluents which makes it readily available in the environment. Release of cyanide into water bodies primarily affects the aquatic organisms which in turn affect terrestrial organisms via food-chain (Mustafa *et al.*, 2015).

Cadmium is known to be heavy metal that is considered to be very toxic due to the fact that its elimination half life is long and it has been known to cause damage to a wide range of organs. Cadmium and cyanide are environmental pollutants (especially aquatic environment) resulting from industrial and human activities (Jaszczak, 2017; Akpor *et al.*, 2014; Osobamiro 2014; Chao *et al.*, 2010). They act as toxicants and can simultaneously occur in the environment (Redman and Santore, 2012). The level of these toxicants in aquatic environment is however on the increase. Contact of these toxicants by humans is mainly through ingestion and inhalation (Satarug 2018; Asagba 2009; Okafor *et al.*, 2002) and long-term low level effect of cadmium and cyanide via the food-chain may trigger toxicity (Sarkara *et al.*,

2016; Shwetha *et al.*, 2012), thus the release of these toxicants into the environment which results in contamination of water bodies should be closely monitored. Fish and other aquatic organisms are susceptible to varying concentrations of cadmium and cyanide exposure and these aquatic organisms have an ability to accumulate cadmium in their tissues far above the aquatic concentrations (Perera *et al.*, 2105) and cyanide which does not bio accumulate is rapidly detoxified (Ramzy, 2014). Fish becomes an important medium of transfer of toxicants in the food chain and this makes it a good bio-indicator (Omar *et al.*, 2014).

The kidney functions as a key organ for the elimination toxins from the body. It processes the blood delivered to it through filtration, reabsorption, secretion and excretion in the form of urine. Alterations in kidney function leads to renal disease which is as a result of damages caused by toxins. Kidney or renal failure (insufficiency) is characterized by a decrease in one or several kidney functions, first the urine concentrating ability, then the elimination of small-molecular-weight molecules from the plasma (DiBartola, 2005).

Although, the toxicity of these two toxicants to wide variety of organs including the kidney have been widely investigated in different countries. However, there is a dearth of knowledge on their combined effects on the kidney. Hence the need for this study

Materials and Methods

Treatment of Fish and Compounding of diet

Sixty Fishes were used for this study. The fishes were assigned into various Groups as follows: Groups A (control) – These were kept in fresh water.

Groups B (Cyanide only) – These were kept in water containing with 0.4mg/L of cyanide.

Groups C (Cadmium only) - These were kept in water containing with 0.4mg/L of cadmium.

Groups D (Cyanide + Cadmium) - These were kept in water containing with 0.4mg/L of both cadmium and cyanide respectively for 28 days.

The water for each Group was changed every day and re-contaminated, this lasted for 4weeks. After which the fishes were killed, oven dried

and used as a source of Protein to compound the animal diet.

Treatment of rats.

Twenty healthy Wistar strain male albino rats were divided into four groups with five rats in each group.

Group 1 (control) – Rats were given control compounded diet.

Group 2 (cyanide) – Rats were given with the cyanide-contaminated compounded diet.

Group 3 (cadmium) – Rats were given cadmium-contaminated compounded diet.

Group 4 (cyanide + cadmium) – Rats were given cyanide + cadmium-contaminated compounded diet. The experiment was for 28days and allowed water was given adlibitum throughout the experimental period.

Collection of tissues

At the end of the 28 days exposure period, the animals were weighed and fasted 12 hours prior to sacrifice and they were sacrificed by cervical decapitation. Laparotomy was carried out to expose the internal organs and blood samples were collected by heart puncture using hypodermic syringe and needle and the sample collected was placed in a plain tube. The kidney was quickly excised, weighed and stored in a container and placed in a refrigerator at 4°C awaiting homogenization.

Preparation of samples

Tissues (0.5g) were homogenized using porcelain mortar and pestle in 4.5ml of 0.1M phosphate buffer (pH 7.4). The homogenate obtained was then placed on a plane tube and centrifuged at 4000rpm for 15 minutes and the supernatant was collected and used for assay. The blood sample collected in plain tubes are centrifuged at 4000rpm for 15 minutes and the serum is obtained is used for biochemical assay. The tissue supernatant and the serum collected are stored in the refrigerator at 4°C prior to use for assay. A section of the kidney was preserved in a universal tube containing 10 % formalin and these tissues used for histopathological studies. Also, samples of the liver and kidney collected were digested in a tube containing 2ml of nitric acid and 3ml of distilled water. The digested samples were homogenized and it was facilitated by heating at 100°C.

Renal indices

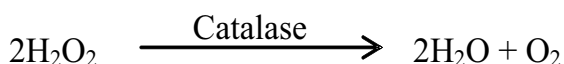
Assay for Serum Urea Activity (Weatherburn, 1967). The absorbance of the sample and standard was read against the blank at 546nm. Assay for Serum Creatinine Activity (Bartels and Bohmer, 1972). The absorbance of the sample was read at 492nm.

Determination of Superoxide dismutase (SOD) activity

The assay for SOD is an indirect method which is based on the inhibitory effects of SOD in the initial rate of epinephrine autooxidation which is derived from the reaction proposed by Misra and Fridovich (1972) for the base catalyzed autooxidation of epinephrine.

Determination of Catalase activity

The method of Rani *et al.* (2004) was adopted for the assay of catalase activity. Catalase breaks down H₂O₂ directly into water and oxygen. The decrease in H₂O₂ concentration may be monitored spectrophotometrically.



Determination of lipid peroxidation (MDA)

The assay for lipid peroxidation (LPO) was done by the method of Niehius and Samuelsson (1968). Malondialdehyde and other thiobarbituric acid reactive species were estimated by their reactivity with thiobarbituric acid (TBA) in acidic condition to generate a pink coloured chromophore which was read at 535 nm in a spectrophotometer.

Determination of reduced glutathione (Moron *et al.*, 1979)

This method for estimation of total reduced glutathione is based on the development of

yellow colour, when dithionitro bis benzoic acid (DTNB) is added to the compounds containing sulphhydryl groups.

Determination of glutathione peroxidase activity (Ellman, 1959)

Glutathione peroxidase catalyses the following reaction:



Glutathione was measured by its reaction with DTNB (5,5'-dithionitro bis-2-benzoic acid) to give a compound that absorbs at 412 nm.

Histology of the Kidney

This was as recommended by Carleton (1967). De-wax sections of the slides were immersed in xylene (2 min) and were hydrated in descending grade of alcohol (absolute, 90%, 70%, 70% for 2 min each). The slides were washed in distilled water (3 min) and stained in haematoxylin for 15 min. After which they were rinsed in tap water for 5 minutes. The slides were then differentiated in 1% acid alcohol (99 ml of 70% alcohol in 1ml of HCl) for 2 min and then washed in running tap water for 5 minutes. The slides were stained in eosin for 3 min and rinse in water for 3 minutes. The slides were dehydrated in ascending grades of alcohol (70%, 90%, and absolute). After which the slides were cleared in xylene and mounted in DPX (DistrenePolystene and Xylene). The stained tissue images were captured using a digital microscopic eyepiece, 'SCOPETEK DCM 500, 8.0 MEGA PIXEL' connected to the USB port of a computer. The stained slides were interpreted with the aid of a light microscope and analyzed for both histological and morphological changes across the groups.

Table 1. Food Chain Borne Effect of Cyanide and Cadmium on Body weight and Organ to body Weight ratio of Rat

Group	A	B	C	D
Bodyweight gain	69.39±8.94 ^a	58.42±7.61 ^b	46.74±12.60 ^c	48.37±10.87 ^c
Kidney/body weight gain	0.59±0.02 ^a	0.64±0.04 ^b	0.59±0.03 ^a	0.63±0.03 ^b

A- Control, B-cyanide contaminated diet, C-cadmium contaminated diet, D- cyanide + cadmium contaminated diet. Values are expressed in mean ± SEM (standard error of mean). Value not sharing same superscript in same row differs significantly at p<0.05

Table 2. Food Chain Borne Effect of Cyanide and Cadmium on Serum renal indices of Rat

Group	A	B	C	D
Urea (mg/dl)	13.31±1.18 ^a	18.55±3.05 ^b	18.41±2.42 ^b	21.57±1.33 ^c
Creatinine (mg/dl)	0.83±0.10 ^a	1.43±0.11 ^b	1.52±0.06 ^b	1.13±0.04 ^c

A- Control, B-cyanide contaminated diet, C-cadmium contaminated diet, D- cyanide + cadmium contaminated diet. Values are expressed in mean ± SEM (standard error of mean). Value not sharing same superscript in same row differs significantly at $p < 0.05$

Table 3. Food Chain Borne Effect of Cyanide and Cadmium on Antioxidant Activity in the Kidney of Wistar Rat

Group	A	B	C	D
SOD (unit/gtissue)	34.06±0.40 ^a	32.11±0.72 ^b	33.90±0.67 ^a	32.46±0.51 ^a
LPO (unit/gtissue)	0.85±0.71 ^a	1.15±0.24 ^b	1.54±0.23 ^c	1.01±0.11 ^b
CAT (unit/gtissue)	60.89±4.56 ^a	48.46±7.16 ^b	44.35±5.35 ^c	42.91±2.78 ^c
GST (unit/gtissue)	15.99±2.19 ^a	11.89±0.99 ^a	12.89±1.31 ^a	15.58±1.32 ^a
GPX (unit/gtissue)	34.73±11.55 ^a	46.13±8.54 ^b	57.80±1.90 ^c	55.90±12.57 ^c
GSH (unit/gtissue)	31.27±4.54 ^a	44.48±6.39 ^b	57.54±4.54 ^c	53.97±1.33 ^c
GR (unit/gtissue)	37.17±2.08 ^a	20.34±2.92 ^b	26.31±2.08 ^c	24.69±0.61 ^c

A- Control, B-cyanide contaminated diet, C-cadmium contaminated diet, D- cyanide + cadmium contaminated diet. Values are expressed in mean ± SEM (standard error of mean). Value not sharing same superscript in same row differs significantly at $p < 0.05$. SOD=Superoxide Dismutase, LPO= Lipid Peroxidation, CAT= Catalase, GST= Glutathione-S-Transferase, GPX= Glutathione Peroxidase, GSH= Reduced Glutathione, GR= Glutathione Reductase

Histology of The Kidney

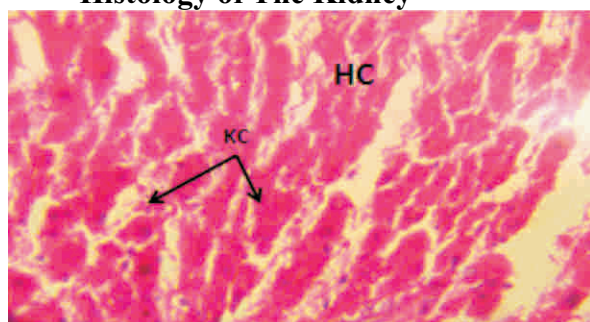


Figure 1: Control group photomicrograph of kidney section showing normal glomerulus (G) Bowman's capsule (BC), and proximal tubules (PT). H&E x400

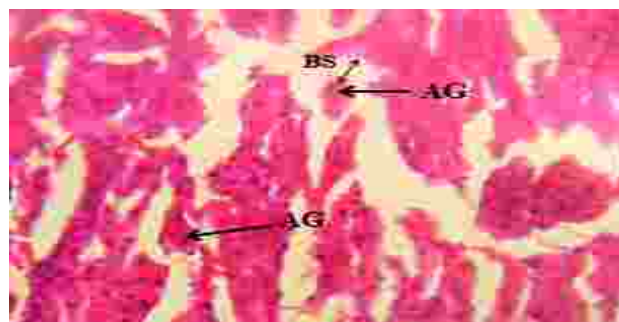


Figure 2: Cyanide contaminated group photomicrograph of rats kidney section showing atrophy glomerulus (AG) and increase Bowman's capsule space (BS).H&E x400.



Figure 3: The cadmium contaminated group photomicrograph shows destruction of glomerulus (G) and acute tubular necrosis (N) and Bowman 's capsule space (BS). H&E x400

Effect of Food Chain Borne Exposure to Cyanide and Cadmium on Body and Organ Weight of Rat

Table 1 shows the effect of food chain borne exposure to cyanide and cadmium on body weight and organ to body weight ratio. The percentage body weight gain of all intoxicated Groups rats fed with cyanide and cadmium contaminated diet both singly and in combination was significantly decreased when compared with control. There was no significance difference ($p > 0.05$) observed in the percentage liver/body weight ratio in rats fed cadmium contaminated diet relative to control. However, a significant increase ($p < 0.05$) observed in the percentage kidney/body weight ratio in rats fed with cyanide-contaminated diet alone and those fed a combination of cyanide and cadmium contaminated diet compared with control. However, no significant difference was observed in the kidney/body weight ratio in rats fed with cadmium-contaminated diet relative to control.

Effect of Food Chain Borne Exposure to Cyanide and Cadmium on Serum Renal Indices of Rat

Table 2 shows the effect of a food chain borne exposure to cyanide and cadmium on serum renal indices of rat. There was a significant increase ($p < 0.05$) in urea level in the groups fed with cyanide and cadmium contaminated diet compared with control. Also, a significant

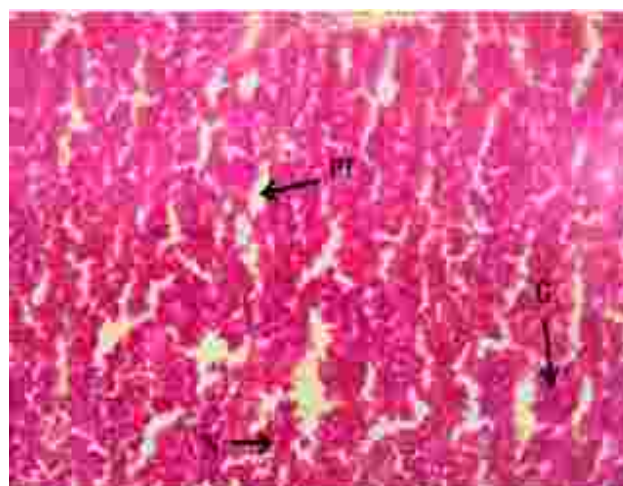


Figure 4: Cyanide and cadmium contaminated group photomicrograph of kidney section showing regeneration of proximal tubules (PT), glomerulus (G) and mild tubular necrosis (N). H&E x400

increase was recorded in the creatinine level in the groups fed with cyanide and cadmium contaminated diet with respect to control group.

Effect of Food Chain Borne Exposure to Cyanide and Cadmium on Antioxidant enzymes in the Kidney of Rat

The effect of food chain borne toxicity of cyanide and cadmium on the kidney of Wistar rat is shown in Table 3. A significant decrease ($p < 0.05$) in superoxide dismutase activity (SOD) and Catalase (Cat) was indicated in the kidney of rats fed with mixture of Cyanide and Cadmium contaminated diet compared when compared with control. However, this decrease was not synergetic when compared with Group B and C given Cyanide and cadmium only respectively. A significant increase ($p > 0.05$) was recorded in the Lipid peroxidation (LPO) level, Glutathione peroxidase (GPX), Glutathione (GSH) and Glutathione Reductase (GR) activity in kidney of rat fed with a mixture of cyanide and cadmium contaminated diet when compared to the control. Again, this increase was not synergistic when compared with Group B and C given Cyanide and cadmium only respectively.

Histological assessment

The histological findings in the kidney of male Wister rats exposed to cyanide and cadmium contaminated diet are shown in the figures below. Figure 1 illustrates the photomicrograph of kidney of control group with normal

glomerulus. Figure 2 shows the photomicrograph of kidney of cyanide contaminated group with acute tubular necrosis. Figure 3 display the photomicrograph of kidney of cadmium contaminated group showing the destruction of glomerulus and acute tubular necrosis. Figure 4 illustrate the photomicrograph of kidney of cyanide and cadmium contaminated group showing normal glomerulus and acute tubular necrosis.

Discussion

The consumption of cadmium and cyanide contaminated diet may manifest in various forms such as physiological and/or biochemical changes. In the present study, results were presented to show the nephrotoxic effect that can emanate following the intake of cadmium and cyanide contaminated diet by rat. Both contaminants have deleterious effect on organ weight and body weight gain of rat and these changes have often been used as an assessment tool of toxicity of these contaminants (Timbrell, 1991).

Serum urea and creatinine levels are key biomarkers of renal dysfunction. In this study, creatinine and urea were estimated in the serum to assess renal globular function. A significant increase was recorded in serum urea level of rat following exposure to cadmium and cyanide tainted diet with the prominent increase observed in the co-exposed tainted diet group indicating that both toxicants has synergistic effect on urea level. However, using urea alone is not a preferred marker for renal function because it is influenced by factors such as a high protein diet, variables in protein synthesis, and hydration status. Creatinine activity is less affected by diet and is a more suitable indicator for renal function. (Jose, 2014).

The increased activity of creatinine in the serum of rat exposed to cadmium and cyanide singly confirms earlier studies which were carried out on rats (Kadiri, 2018; Okolie and Osagie 2000). In addition, a significant increase in creatinine level was also indicated in Group D given the combined toxicant in their diet; however, the increase was not synergistic. The alterations in the level of serum renal biomarkers represent

kidney damage caused by toxicity of xenobiotic has previously been reported in literature (Gabr *et al.*, 2017; Bekheet *et al.*, 2011; Okolie and Osagie 2000). Also, these toxicants induce tubular necrosis or loss of the brush border which causes damage in the small tubule of kidney (Wang *et al.*, 2009). Gabr *et al.* (2017) also reported that high blood urea initiated by cadmium toxicity may be correlated to disorder in protein catabolism as a result of increase in the synthesis of arginase enzyme involved in urea production and glomerular functional disturbances such as lower filtration rate which result in poor creatinine clearance in rat exposed to cadmium. The result was further confirmed by the histological studies in Figure 1-4. This indicated that the extent of damage caused by the mixed toxicant was less than that caused by the individual toxicants, particularly cadmium.

Some oxidative stress enzymes such as superoxide dismutase (SOD), catalase (CAT), glutathione peroxidase (GPx), glutathione-*s*-transferase (GST) and glutathione (GSH) and the levels of MDA, an end product of lipid peroxidation, were examined in the present study to ascertain the effect of cadmium and cyanide contaminated diet induced oxidative stress in the kidney of rat. Studies have shown that the activity of antioxidant enzymes acts in two diverse ways during oxidative stress. Firstly, at the onset of stress this activity increases, while on the long run, it is reduced due to the enormous production of free radicals. This decline is the result of impairment to the molecular mechanism that is necessary to induce these enzymes (Casalino *et al.*, 2002).

In this study, a significant decrease was observed in the SOD and CAT activity in the kidney of rats following exposure to cadmium and cyanide tainted diet (singly) and this finding is in line with earlier studies in Cyanide (Kadiri and Asagba, 2019; Petkovic *et al.*, 2018) and in cadmium (Elmallah *et al.*, 2017). In addition, significant decrease was observed in the SOD and CAT activity in the kidney of rats following exposure to a mixed cadmium and cyanide tainted diet, However the effect was not synergistic when compared with Groups B and C given cyanide and Cadmium only. Superoxide

anions is generated in the cells due to exposure to xenobiotic and it initiates the active role of SOD which terminates the harmful radicals in cell and this might lead to the decline in SOD activity, while CAT is a tetrameric enzyme, they are important in relieving oxidative stress. An increase in MDA concentrations in Groups B and C exposed to cyanide and cadmium tainted diets singly is in accordance with earlier studies (Kadiri and Asagba 2019; Petkovic et al., 2018) on cyanide and in cadmium (Jafarpour *et al.*, 2017). An increase in the activity of MDA in the kidney in all the groups exposed to the toxicant suggests that the antioxidant defense system of the rats was damaged due to oxidative stress. Also, the increased LPO may be due to decreased activities of SOD and CAT, the free radical scavenging enzymes which may result to organs disruption. The observed outcomes are in accordance with previously stated studies by Mossa *et al.*, 2015; Cosic *et al.*, 2007 which shows that there was a significant decline in antioxidant enzymes and increase in MDA levels in rats exposed to xenobiotic.

GST is a GSH-dependent enzyme that catalyzes the conjugation reaction of xenobiotic. Xenobiotic can be easily transported or excreted by GST via conjugation reaction which makes it hydrophilic. The result observed in this study showed that the concentration of GST decreased in the kidney of rats exposed to cyanide and cadmium polluted diet. This result is in agreement with the work reported by Akcha *et al.* (2000) who reported a decrease in GST activity in mussels *Mytilus galloprovincialis* exposed to xenobiotic. On the other hand, an increase in GST activity in organisms exposed to xenobiotic was recorded by several reports (Ensibi and Daly Yahia, 2017; Emadeldeen 2014). However, no significant change in GST activity in *Daphnia magna* exposed to cadmium was reported in a previous report (Jamec *et al.*, 2007). Thus, the variation in GST response might be due to species or duration exposed to xenobiotic (Ensibi and Daly Yahia, 2017). It was observed in this study that GPx activity increases significantly in the kidney of rats exposed to contaminated diet while no change was observed in the liver. This increase in GPx activity is attributed to ROS accumulation as a result of the detoxification of

H₂O₂ and this finding are in line with earlier report (Ensibi and Daly Yahia, 2017).

GSH is an important antioxidant which is responsible for controlling intracellular levels of lipid peroxidation and the detoxification of xenobiotic. Observations from this study show that there was a significant elevation in GSH activity in liver and kidney of exposed animals. This increase indicates oxidative stress induced in the animals following exposure to contaminated diet. This observation is in agreement with the work of Shams and Ahmad (2011) who also reported elevated GSH activity in tissues of rats exposed to toxicant. GR which is also a vital antioxidant enzyme catalyzes the regeneration of GSH from GSSG thus making it an essential cellular antioxidant protector. Results from the present study shows that the GR activity in the liver of exposed rats was significantly increased while a significant decrease was observed in the kidney. Studies have shown that the susceptibility of species exposed to xenobiotic in addition to the intensity of the stress induced as well as the duration exposed to the stress is responsible for inhibited or elevated activities of antioxidant enzyme (Ensibi and Daly Yahia, 2017).

Conclusions

The findings of this study indicate that exposure of a mixture of cadmium and cyanide via the food chain causes damage to the kidney of the rats. However, their combined effect was antagonistic.

References

- Akcha, F., Izuel, C., Venier, P., Budzinski, H., Burgeot, T. and Narbonne, J.F. (2000). Enzymatic biomarker measurement and study of DNA adduct formation in benzo(a)pyrene contaminated mussels *Mytilus galloprovincialis*. *Aquatic Toxicology*; 49: 269–287.
- Akpor, O.B., Ohiobor, G.O and Olaolu, T.D. (2014). Heavy metal pollutants in wastewater effluents: Sources, effects and remediation. *Advances in Bioscience and Bioengineering*; 2(4): 37-43.
- Asagba, S.O. (2009). Role of diet in absorption

- and toxicity of oral cadmium- A review of literature. *African Journal of Biotechnology*; 8(25):7428-7436.
- Bartels, H. and Bohmer, M. (1972). *Clinical Chemistry Acta*; 37:193.
- Bekheet, S.H., Awadalla, E.A., Salman, M.M. and Hassan, M.K. (2011). Bradykinin potentiating factor isolated from *Buthus occitanus* venom has a protective effect against cadmium induced rat liver and kidney damage. *Tissue Cell*; 43: 337-343.
- Casalino, E., Calsaretti, G., Sblano, C., Landriscina, C. (2002). Molecular inhibitory mechanisms of antioxidant enzymes in rat liver and kidney by cadmium. *Toxicology*; 179: 37–50.
- Chao, Y., Qicong, L., Sha, Y., Jing, L., Zhongda, C and Zhenyu, P. (2010). Cadmium contamination in various environmental materials in an industrial area, Hangzhou, China. *Chemical Speciation and Bioavailability*; 22 (1) 35-42.
- DiBartola, S.P. (2005). Renal disease: clinical approach and laboratory evaluation. In “Textbook of Veterinary Internal Medicine” (Ettinger, S.J. and Feldman, E.C. Eds.), 6th ed Elsevier Saunders, St. Louis, MO.:1716 – 1730.
- Elmallah, M., Elkhadragey, M. F., Al-Olayan, E. M. and Abdel Moneim, A. E. (2017). Protective Effect of *Fragaria ananassa* Crude Extract on Cadmium-Induced Lipid Peroxidation, Antioxidant Enzymes Suppression, and Apoptosis in Rat Testes. *International journal of molecular sciences*, 18(5), 957
- Ellman, G.L. (1959). Tissue sulfhydryl groups. *Archives of Biochemistry and Biophysics*; 82:70–77.
- Emadeldeen, H.M. (2014). Biochemical response of the cyclopoida copepod *Apocyclops borneoensis* exposed to nickel. *Jordan Journal of Biological Science*; 7 (1): 41–47.
- Ensibi, C., Daly Yahia, M. N. (2017). Toxicity assessment of cadmium chloride on planktonic copepods *Centropages ponticus* using biochemical markers. *Toxicology Reports*; 4:83–88.
- Ezedom, T and Asagba, S.O. (2016). Effect of a controlled food-chain mediated exposure to cadmium and arsenic on oxidative enzymes in the tissues of rats. *Toxicology Reports*; 3:708-715.
- Fetoui, H., Makni, M., Garoui, El-M., Zeghal, N. (2010). Toxic effects of lambda-cyhalothrin, a synthetic pyrethroid pesticide, on the rat kidney: Involvement of oxidative stress and protective role of ascorbic acid. *Experimental Toxicology and Pathology*; 62: 593-599.
- Gabr, S.A Alghadir, A.H. Ghoniem, G.A. (2017). Biological activities of ginger against cadmium-induced renal toxicity. *Saudi Journal of Biological Science*; 26: 382–389.
- Grotto, D., Valentini, J., Serpeloni, J.M., Monteiro, P.A.P., Latorraca, E.F., deOliveira, R.S. and Barbosa Jr. F. (2011). Evaluation of toxic effects of a diet containing fish contaminated with methyl mercury in rats mimicking the exposure in the Amazon riverside population, *Environmental Research*; 111: 1074–1082.
- Jafarpour, D., Shekarforoush, S. S., Ghaisari, H. R., Nazifi, S., Sajedianfard, J., & Eskandari, M. H. (2017). Protective effects of synbiotic diets of *Bacillus coagulans*, *Lactobacillus plantarum* and inulin against acute cadmium toxicity in rats. *BMC Complementary and Alternative Medicine*; 17(1): 291.
- Jaszczak, E., Polkowska, Z., Narkowicz, S and Namieśnik, J. (2017). Cyanides in the environment, analysis, problems and challenges. *Environmental Science Pollution and Research*; 24: 15929–15948.
- Jemec, A., Drobne, D., Tisler, T. et al., (2007). The applicability of acetyl cholinesterase and glutathione S-transferase in *Daphnia magna* toxicity test. *Comparative Biochemistry and Physiology Part C*; 144: 303–309.
- Kadiri, H. E (2017). Protective effect of *vernonia amydalina* (bitter leaf) extract on rats exposed to cyanide poisoning: *Biokemistri*; 29 (3), 126-131.
- Kadiri, H.E. and Asagba, S.O. (2019). The chronic effects of cyanide on oxidative stress indices in the chicken (*Gallus domesticus*). *The Journal of Basic and Applied Zoology*. 80(30):1-11

- Maranesi, M., Zambonin, L., Tolomelli, B., Cabrini, L., Bochicchio, D. (2004). Effects of different dietary amounts of LCPUFA n3 and vitamin B 6 on lipid composition and antioxidant defences in rat kidney. *Journal of Nutritional Biochemistry*; 15: 396-401.
- Misra, H.P. and Fridovich, I. (1972). The role of superoxide anion in the autoxidation of epinephrine and a simple assay for superoxide dismutase. *Journal of Biological Chemistry*; 247: 3170.
- Moron, M.S., Depierre, J.W. and Mannervik, B. (1979). Levels of glutathione, glutathione reductase and glutathione S-transferase activities in rat lung and liver. *Biochimica et Biophysica Acta*; 582: 67-78.
- Mustafa, K., Yusuf, S., Kazim U., Muhammet, D., Sayit, A., Irfan, Y and Halil, I. (2015). Effect of sodium cyanide on Antioxidant enzyme activities and lipid peroxidation in some tissues of Mirror Carp (*Cyprinus carpio*). *Pakistan Journal of Zoology*; 47(6):1777-1782.
- Niehius, W.G. and Samuelsson, B. (1968). Formation of phospholipid arachidonate during microsomal lipid peroxidation. *European Journal of Biochemistry*; 6: 126 - 130.
- Okafor, P.N., Anyanwu, V.O., Onyema, H.O. (2010). The effects of cassava cyanide on the antioxidant (Glutathione) status and some clinically important enzymes of rats. *Journal of Pharmacology and Toxicology*; 5(7): 389-395.
- Okafor, P.N., Okorowkwo, C.O and Maduagwu, E.N. (2002) Occupational and dietary exposures of humans to cyanide poisoning from large-scale cassava processing and ingestion of cassava foods. *Food Chemistry and Toxicology*; 40:1001-1005.
- Okolie, N. P., Osagie, A. U. (2000). Differential effects of chronic cyanide intoxication on heart, lung and pancreatic tissues. *Food Chemistry and Toxicology*; 38:543-548.
- Okolie, N.P., Iroanya, C.U. (2003). Some histologic and biochemical evidence for mitigation of cyanide induced tissue lesions by antioxidant vitamin administration in rabbits. *Food Chemistry and Toxicology*; 41: 463-469.
- Omar, W.A., Saleh, Y.S. and Marie, M.A. (2014). Integrating multiple fish biomarkers and risk assessment as indicators of metal pollution along the Red Sea coast of Hodeida, Yemen Republic. *Ecotoxicology Environmental Safety*; 110:221-231.
- Osobamiro, M.T. (2012). Determination of the Concentration of Total Cyanide in Waste Water of a Tobacco Company in Southwestern Nigeria. *Journal of Applied Science and Environmental Management*; 16(1): 61 - 63.
- Petkovic, A.M., Jakovljevic, V.L., Bradic, J.V., Jeremic, J.N., Jeremic, N.S., Nikolic Turnic, T. R., Zivkovic, V.I. (2018). The Effects of Potassium Cyanide on the Functional Recovery of Isolated Rat Hearts after Ischemia and Reperfusion: The Role of Oxidative Stress. *Oxidative Medicine and Cellular Longevity*; 2018: 5979721.
- Perera, P., Kodithuwakku, S.P., Sundarabarathy, T.V and Edirisinghe, U. (2015). Bioaccumulation of Cadmium in Freshwater Fish: An Environmental Perspective. *Insight Ecology*; 4(1): 1-12.
- Ramzy, E.M. (2014). Toxicity and stability of sodium cyanide in fresh water fish Nile tilapia. *Water Science*; 28: 42-50.
- Rani, A., Kumar, A., Lal, A. and Pant, M. (2014). Cellular mechanisms of cadmium-induced toxicity: a review. *International Journal of Environmental Health Research*; 24 (4): 378-399.
- Redman, A and Santore, R. (2012). Bioavailability of cyanide and metal-cyanide mixtures to aquatic life. *Environmental Toxicology and Chemistry*; 31 (8):1774-1780.
- Sarkara, T., Masihul Alama, M., Parvina, N., Fardousb, Z., Chowdhuryb, A.Z., Hossainc, S., Haqued, M.E and Biswas, N. (2016). Assessment of heavy metals contamination and human health risk in shrimp collected from different farms and rivers at Khulna-Satkhira region, Bangladesh. *Toxicology Report*; 3: 346-350.
- Satarug, S. (2018). Dietary cadmium intake and its effects on kidneys. *Toxicology*; 6 (15): 1-23.
- Shams, T., Ahmad, M. (2011). Some enzymatic/non enzymatic antioxidants as potential biomarkers of trichloroethylene,

heavy metals mixture and ethyl alcohol in rat tissues. *Environmental Toxicology*; 26(2): 207-216.

Shwetha, A., Hosetti, B.B and Dube, P.N. (2012). Toxic Effects of Zinc Cyanide on Some Protein Metabolites in Fresh water fish, *Cirrhinus mrigala* (Hamilton). *International Journal of Environmental Research*; 6(3):769-78.

Timbrell, J.A. (1991). Principles of Biochemical

Toxicology. 2nd Edition. Taylor and Francis, London.

Wang, L., Chen, D., Cao, J., Liu, Z. (2009). Protective effect of N-acetylcysteine on experimental chronic cadmium nephrotoxicity in immature female rats. *Human and Experimental Toxicology*; 28: 221-229.

Weatherburn, M. W. (1967). Urea Concentration Estimation in samples. *Annals of Chemistry*; 39: 971.

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