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# Hepatoprotective Effect Of The Aqueous Extract Of *Mucunapruriens* (Velvet Beans) In Nickel Chloride Induced Toxicity In Albino Rats

Adegbola M. V.[1*], Dada F. A.[1]
[1]*Science Laboratory Technology Department (Biochemistry Unit), Federal Polytechnic Ede, Nigeria.*

***Abstract:***
***Background****:Velvet beans (Mucunapruriens) is a tropical leguminous plant that belongs to Mucuna genus, the Fabaceae family. Various studies have reported the bioactivity of Mucunapruriens, such as increasing the sexual activity of normal male rats, hepatoprotective activity and its use in the treatment of diabetes and hypertension.*
***Materials and Methods****: In this study, its hepatoprotective ability was investigated in rats induced with $NiCl_2$ toxicity. Alanine aminotransferase (ALT) activity, aspartate aminotransferase (AST) activity and arginase activity were determined in rats treated with aqueous extracts of Mucunapruriens (MPE). Lipid peroxidation was also determined by measuring malondialdehyde (MDA) levels in the liver homogenate of $NiCl_2$-induced rats.*
***Results****: It was observed that alanine aminotransferase and aspartate aminotransferase (AST) activities were elevated in rats induced with $NiCl_2$. These activities were however significantly reduced in the treatment groups, with the group treated with 200mg/kg bodyweight MPE showing greater activity. Arginase activity in the liver homogenate of Nickel chloride induced toxicity rats was elevated. This was significantly mediated with in MPE treatment groups. The aqueous extract of Mucunapruriens only groups (100mg/kg and 200mg/kg bodyweight) showed lower activity of arginase than even the control group. Malonaldehyde levels (MDA) was significantly increased in the rats administered with nickel chloride. However, treatment with both 100mg/kg and 200mg/kg bodyweight of Mucunapruriens significantly reduced this($p<0.05$). The values obtained were comparable to that of the standard Quercetine.*
***Conclusion:****This study has helped to establish the hepatoprotective potential of Velvet beans and validate its use in traditional herbal medicine*.
***Key Word****:Hepatoprotective, Lipid peroxidation,Quercetin,Mucunapruriens, Nickel chloride.*

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## I. Introduction

Liver toxicity simply refers to damage done to the liver caused by excessive consumption of alcohol, persistent use of medications/drugs, long exposure to chemicals/solvents, and indiscriminate use of herbal supplements (). Studies had shown hepatotoxicity in rats exposed to nickel chloride and elevation in plasma alanine aminotransferase (ALT) and plasma aspartate aminotransferase (AST)[1,2]. Several factors such as elderly people, female gender, abuse of drugs, pregnancy, and constant alcoholism have been implicated as the causes of hepatotoxicity [3]. Nickel is described as a substance that is toxic to the liver, blood, kidney, and immune system[4].It also forms a complex with adenosine triophosphate, peptides, deoxyribonucleic acid, and amino acids[5]. Nickel has been reported to trigger oxidative stress in liver via generation of free radicals which eventually damage hepatocellular cells[6]. However, plants rich in phenolic compounds and antioxidative properties have been suggested to combat oxidative stress and protect liver cells from the attack of free radicals[7]. *Mucunapruriens* (MPE) is a tropical legume usually found and cultivated in Africa, it has also been used from time immemorial in the traditional medicine to treat various diseases. MPE has been described as a plant embedded with numerous pharmacological and antioxidative properties capable of eliminating activities of oxidative stress[8]. There is limited scientific information on the hepatoprotective potentials of MPE in nickel chloride induced hepatotoxicity, as such, this study tends to investigate hepatoprotective properties of MPE in nickel chloride induced toxicity in rats' liver.

## II. Material And Methods

This experiment was carried out in the Biochemistry laboratory, Department of Science Laboratory Technology Department, Federal Polytechnic Ede, Nigeria.

**Chemicals**

Thiobarbituric acid (TBA), acetyltiocholine and butrylthiocholine iodide, ammonium ferrous sulfate, potassium iodide, sodium potassium tartrate, copper sulfate, glacial acetic acid, trichloroacetic acid, potassium dichromate, hydrogen peroxide (H2O2), hydrochloric acid, sulfuric acid, sodium hydroxide, quercetin, benzylamine, semicarbazide, ethanol, were purchased from Sigma (St Louis, MO, USA). All other chemicals used were of analytical grade and obtained from British Drug Houses (Poole, Dorset, UK). Also, ALT and AST Kits were purchased from Randox Laboratories Limited, Crumlin County Antrim, United Kingdom.

**Care of animals**

All the experimental animals received humane care, according to the criteria outlined in the Guide for the Care and Use of Laboratory Animals prepared by the National Academy of Science and published by the National Institutes of Health [9]. The ethics regulations were followed in accordance with national and institutional guidelines for the protection of the animals' welfare during experiments according to the Public Health Service [9].

**Experimental design**

Fourty-eight (48) adult male rats weighing between 180 and 200 g were randomly divided into 8 groups of 6 animals per group. The animals were kept in wire mesh cages under controlled light cycle (12h light/12h dark); they were fed with commercial rat chow ad libitum and liberally supplied with water.

**Group 1** rats served as the control group and received normal saline orally for 7 days;
**Group 2** rats were given 10 mg/kg of Nickel chloride ($NiCl_2$) solution orally per body weight;
**Group 3** was given 10 mg/kg of $NiCl_2$ solution and 25 mg/kg of Quercetin (QE) orally per body orally
**Group 4** was given 10 mg/kg of $NiCl_2$ solution and 100 mg/kg of Mucuna purine (MPE) extract rally
**Group 5** was administered with 10 mg/kg of $NiCl_2$ solution and 200 mg/kg of Mucuna purine extract rally
**Group 6** rats received 100 mg/kg of MPE extract only
**Group 7** rats received 200 mg/kg of MPE extract only
**Group 8** rats received 25 mg/kg of QE extract only

On 7th day, the rats were sacrificed; the blood sample was collected from the punctured heart of the rats and centrifuged at 3000rpm for 10 minutes. Thereafter, serum was collected and stored in sample bottles for further biochemical analysis. Also, the liver tissue was rapidly removed, rinsed in cold saline and homogenized with 0.1M phosphate buffer (pH 7.4) solution. Thereafter, the homogenate was centrifuged to obtain clear supernatant which was used for biochemical assays.

**Biochemical assays**

Serum alanine aminotransferase (ALT) and aspartate aminotransferase (AST) were analyzed by using commercially available kits from RANDOX Laboratories Ltd, Crumlin County Antrim, UK and the procedures stated therein were strictly followed.

The contents of MDA levels were obtained from the brain homogenate by method described by Ohkawa et al[10]. The arginase activity was determined by the method of Zhang et al.[11].

**Statistical analysis**

All data were expressed as mean ± standard error of mean (S.E.M.). One-way ANOVA was used for the statistical analysis followed by the post hoc Tukey's test; $P < 0.05$ was considered to represent a significant difference using GraphPad prism software, 5.0 version.

## III. Result

Figure 1 revealed the effect of *Mucunapruriens* on the activity in nickel chloride ($NiCl_2$)-induced toxicity in rats' liver. Administration of $NiCl_2$ led to elevation of alanine aminotransferase (ALT) activities in rats that received $NiCl_2$ only compared with normal control rats. However, induced rats treated with 25 mg/kg quercetin (QE) and 100 & 200 mg/kg *Mucunapruriens* (MPE) had reductions in ALT activities with no significant difference ($P > 0.05$) in comparison with the untreated rats.

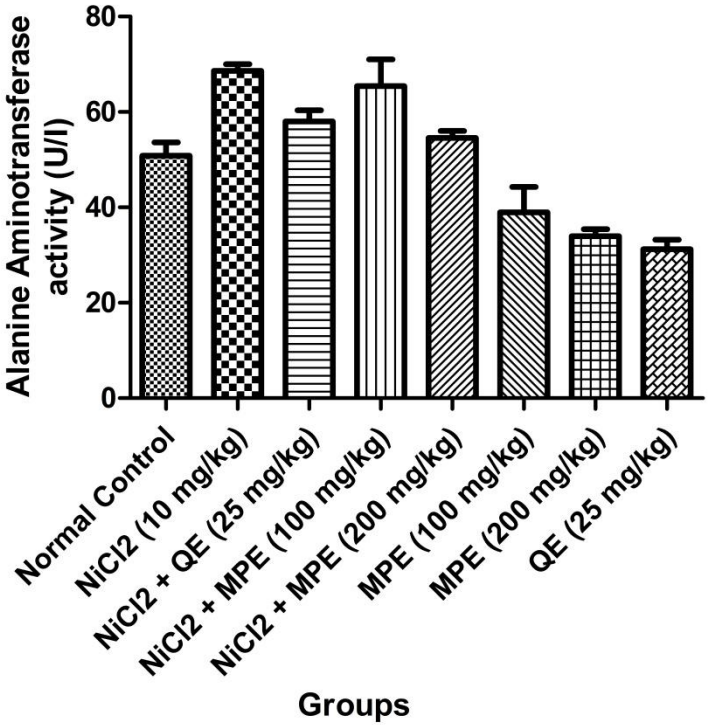


**Figure 1**: Effect of *Mucunapruriens* on alanine aminotransferase (ALT) activity in nickel chloride-induced toxicity in rats' liver. Bars represent mean ± standard deviation of triplicate readings (n = 6). QE= Quercetin; $NiCl_2$ = nickel chloride; MPE= *Mucunapruriens*

As evidenced in figure 2, rats administered with $NiCl_2$ only had significant increase in aspartate aminotransferase (AST) activities when compared with normal control rats. Meanwhile, rats treated with 25 mg/kg quercetin (QE) and 100 & 200 mg/kg *Mucunapruriens* (MPE) had reductions in AST activities with no significant difference ($P > 0.05$) in comparison with the untreated rats.

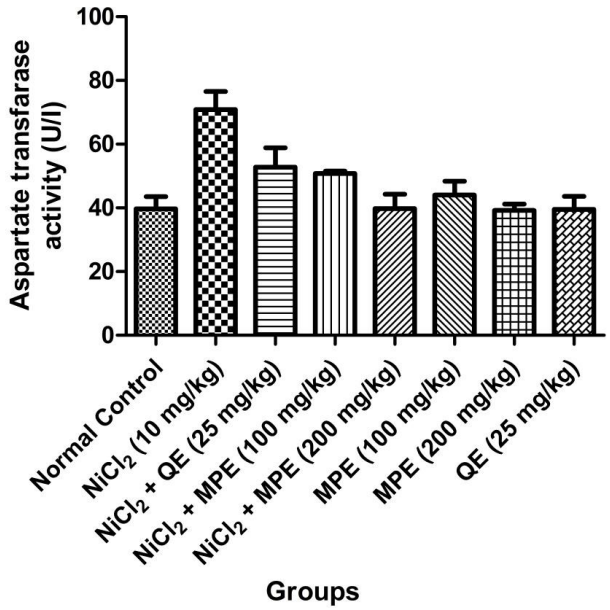


**Figure 2**: Effect of *Mucunapruriens* on aspartate aminotransferase (AST) activity in nickel chloride-induced toxicity in rats' liver. Bars represent mean ± standard deviation of triplicate readings (n = 6). QE= Quercetin; $NiCl_2$ = nickel chloride; MPE= *Mucunapruriens*

Rats that received $NiCl_2$ only had significant high arginase activity, compared with the normal control rats (figure 3) while rats that received $NiCl_2$ + QE (25 mg/kg) and $NiCl_2$ + MPE (100 & 200 mg/kg) had decrease in arginase activity compared with untreated rats ($NiCl_2$10mg/kg).

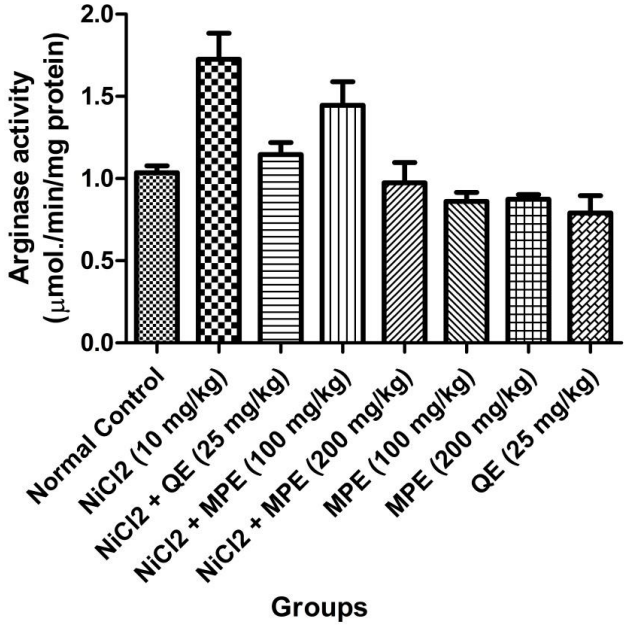


**Figure 3**: Effect of *Mucunapruriens* on arginase activity in nickel chloride-induced toxicity in rats' liver. Bars represent mean ± standard deviation of triplicate readings (n = 6). QE= Quercetin; $NiCl_2$ = nickel chloride; MPE= *Mucunapruriens*

Figure 4 revealed the effect of *Mucunapruriens* on malondealdehyde (MDA) concentration in nickel chloride ($NiCl_2$)-induced toxicity in rats' liver. Administration of $NiCl_2$ led to significant elevation of MDA concentration in rats that received $NiCl_2$ only compared with normal control rats. However, induced rats treated with 25 mg/kg quercetin (QE) and 100 & 200 mg/kg *Mucunapruriens* (MPE) had reductions in MDA) concentration with no significant difference ($P > 0.05$) in comparison with the untreated rats.

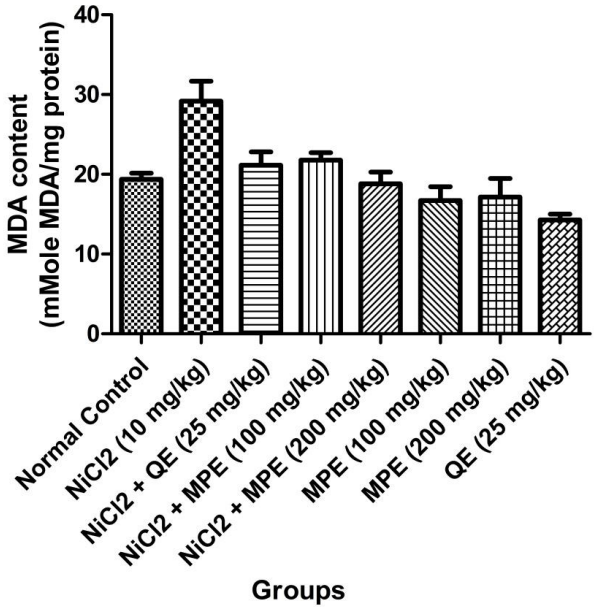


**Figure 4**: Effect of *Mucunapruriens* on malondealdehyde concentration in nickel chloride-induced toxicity in rats' liver. Bars represent mean ± standard deviation of triplicate readings (n = 6). QE= Quercetin; $NiCl_2$ = nickel chloride; MPE= *Mucunapruriens*

## IV. Discussion

Liver biomarkers are biological components used to detect progression of disease in the liver, the most common are alanine aminotransferase (ALT) and aspartate aminotransferase (AST), and abnormal increase in the levels of the biomarkers have been described as the pathogenesis of liver disease. Several studies had reported abnormal increase in ALT and AST levels which were triggered by the induction of nickel chloride[12,13]. Our results from this study confirmed the elevation of AST and ALT in the rats that were induced with nickel chloride, compared with the control rats. This may occur as a result of the activities of oxidative stress, originated by the induction of nickel chloride which may have damaged the liver cell and tissues. However, when the induced rats were treated with quercetin (QE) and *Mucunapruriens* (MPE) 100 and 200 mg/kg, reductions in the biomarkers levels were observed in comparison with the induced/untreated rats. This mechanism of action may be possible due to antioxidative potentials of MPE to eliminate free radicals; this finding reveal the hepatoprotective abilities of MPE.

Arginase is the last step in the urea cycle, it splits L-arginine to form L-ornithine and urea, also reduces L-arginine supply needed for the production of nitric oxide (NO) by NO synthase while malondialdehyde (MDA) is an organic compound and oxidative stress biomarker. Increased activities of arginase and MDA have been implicated in the dysfunction and damage of several organs such as kidney, liver, and central nervous system[14,15]. The findings in our work showed increased activities of arginase and MDA in the nickel chloride induced rats when compared with the normal control rats. But upon treatement of the induced rats with QE and MPE (100 & 200 mg/kg), the elevated arginase activity and MDA level were reversed when compared with the untreated rats. The observed reversals may be due to the phenolic compounds present in MPE which were able to scavenge free radicals in the liver tissue. Our finding is in agreement with the study reported by Lampariello et al[16].

## V. Conclusion

This study has established the hepatoprotective potentials of *Mucunapruriens* by lowering the levels of liver biomarkers (alanine aminotransferase and aspartate aminotransferase) and inhibiting the activities of enzymes linked to hepatotoxicity.

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