# **ASSESSMENT OF RENAL FUNCTION IN DRIVERS CONSUMING ALCOHOL AND NSAID IN OLUYOLE LOCAL GOVERNMENT AREA, IBADAN, OYO STATE**

### **Abstract**

**Background:** There is a strong relationship between alcohol use disorder (AUD) and kidney dysfunction, and it remains a mystery, with little or no research to explain the cause. In this present research, we delve into the impact of alcohol consumption and the use of nonsteroidal anti-inflammatory drugs (NSAID) on renal function and hypertension prevalence among commercial bus drivers, a more vulnerable occupational group.

**Methods:**  This cross-sectional study used commercial bus drivers as participants, and assessed their serum creatinine, urea, potassium, and blood pressure levels. These Participants were grouped based on their alcohol consumption and NSAID use, and statistical analysis was done to determine the associations between these variables and renal function markers.

**Results:** we reported **12.1% of participants to have had a high creatinine level**, although no reasonable difference was seen statistically between alcohol consumers and non-consumers. **(p>0.05)**. Hypokalemia was seen in about **42.4% of participants,** showing a statistically significant deviation from controls **(p<0.001),** underscoring the likelihood of alcohol-induced electrolyte imbalances. **15% of participants had** Hypertension, with **age and BMI (>25 kg/m²) presented as significant risk factors**. Nevertheless, no statistically significant relationship was seen between hypertension and renal function markers **(p>0.05)**.

**Conclusion:** Although we did not see a notable alteration in the serum creatinine levels of alcohol consumer participants, there were a lot of **electrolyte imbalances and hypertension** risk, which highlight the urgent need for frequent health checkups, diet checks, and occupational health interventions among commercial bus drivers. Future research endeavors should make use of **direct GFR measurement and longitudinal designs** to establish causality and identify early markers of alcohol-induced renal dysfunction.

**Keywords:** Alcohol use disorder, renal function, hypertension, potassium imbalance, commercial drivers, NSAIDs

**Introduction**
Each profession has its hazards, with some carrying more risk than others. Among these, driving commercially has been recognized as an occupation that is prone to a high risk of hazards, especially when safety protocols are not well put in place (1). Developing countries such as Nigeria struggle with adherence to occupational health and safety (OHS) regulations. The commercial system of transportation, which is an important component of urban mobility and economic development, suffers from this limitation the most(2). These policies from OHS were originally made to ensure workers are protected physically, mentally, and socially by reducing the risk of occupational hazards. These measures were supposed to be strictly adhered to for commercial bus drivers because they are more vulnerable to physical, psychological, and social risks(3).

The socioeconomic growth of most urban areas depends largely on their Commercial transportation system, because it enhances connectivity, minimizes traffic, and ensures economic productivity(4). Despite how important this is, this sector faces the most considerable challenges, especially when it comes to the health and safety of drivers. Commercial drivers most of the time face risks from the environment that expose them to long-term health risks(5). Nigeria currently faces a menace in its commercial transportation system, which is the usage of psychoactive substances like alcohol, tobacco, and cannabis, among these drivers(6). Regardless of the numerous sensitization programs by the Federal Road Safety Corps (FRSC) and civil society organizations about this, the excessive use of substances remains prominent among Nigerian drivers. There is currently no notable legal framework that establishes blood alcohol concentration (BAC) limits for commercial drivers in Nigeria, subsequently leading to continuous safety and health concerns. A major contributing factor to the problem is the widespread availability of alcohol and other stimulants in motor parks and roadside establishments(6). A study conducted by Ozoh et al (2017 reported more than 3,000 motor parks, kiosks, and eateries along Nigerian highways as spots for alcohol and tobacco consumption among drivers(7). These substances are claimed to be used to fight tiredness and fatigue and regain strength after a long day at work.

Another study by Olaniyi et al 2020 on the behavior of commercial bus drivers while driving in the southwestern part of Nigeria showed that 60% of drivers who were 45 years and below, frequently consume alcohol while driving(8). This and other risk factors worsening the incidence of road accidents in Nigeria include inadequate education, poor road conditions, and bad vehicles. Additionally, drivers mostly take energizers to remain awake during long trips. All these have not only increased the risk of road accidents but also put the health of the drivers at risk of long-term severe health conditions(9).

Although behavioral risks that are linked with substance abuse among commercial drivers have been well-documented, there is limited or no research on the long-term physiological consequences, particularly concerning renal function(6). There are physiological consequences to abusing substances, especially on renal health, leading to further research. The kidneys are very important in maintaining the body's homeostasis, filtering waste products, and ensuring essential body fluids are regulated(10). There has been notable evidence of renal impairment due to exposure to toxic substances such as alcohol and non-steroidal anti-inflammatory drugs (NSAIDs). NSAIDs are mostly used for the relief of pain; they act by inhibiting cyclooxygenase enzymes, thereby hindering the production of prostaglandin, a very important intermediary in renal metabolism(11). Addiction to these substances can lead to difficulty in renal filtration, imbalances of electrolytes, and systemic complications. Due to the ongoing menace of high rates of alcohol and NSAID consumption among Nigerian commercial drivers, there is an urgent need to examine their potential impact on renal health(12). This present study aimed to bridge this gap by investigating key biomarkers of renal function, including plasma urea, creatinine, potassium levels, and glucose, among commercial drivers in Nigeria.

# **MATERIALS AND METHODS**

The following laboratory materials were used for sample collection, processing, and analysis:

* **Sample Collection and Handling**: Cotton wool, disposable hand gloves, sterile needles and syringes, lithium heparin tubes, fluoride oxalate tubes, and plain serum tubes.
* **Laboratory Equipment**: Centrifuge (for plasma separation), spectrophotometer (for biochemical analysis), glucometer (for point-of-care glucose testing), water bath (for incubation), ion-selective electrode (ISE) analyzer (for electrolyte determination), micropipettes (for precise reagent handling), pipette tips, and a refrigerator (for sample storage).

### **Reagents**

* **Creatinine Analysis:** Randox Creatinine Assay Kit (based on Jaffe's reaction)
* **Urea Analysis:** Randox Urea Kit (based on urease-Berthelot reaction)
* **Glucose Analysis:** Randox Glucose Kit (utilizing glucose oxidase-peroxidase reaction)

## **Study Location**

The study was conducted at various motor parks in **Oluyole Local Government Area, Ibadan, Oyo State, Nigeria**. Ethical approval was obtained from the **Ministry of Health, Oyo State,** and informed consent was secured from all study participants before enrollment.

## **Study Design**

It was a **cross-sectional, comparative study** that evaluated renal function among commercial drivers who consume alcohol and NSAIDs, with a control group of non-transport workers.

## **Study Population**

The study recruited a total of **200 participants:**

* **100 commercial drivers** (exposed group)
* **100 non-commercial drivers** (control group)

Both groups were matched for age and gender to minimize confounding variables.

## **Sample Size Calculation**

The sample size was calculated using **Cochran’s formula for a single proportion,** based on an existing prevalence rate of **11.5%** from a study on visual impairment among commercial drivers in Nigeria. The final sample size was adjusted to **200 participants** to account for a **10% non-response rate.** (13)

## **Ethical Considerations**

Ethical approval was obtained from the **Ethical Committee of the Ministry of Health, Oyo State.** Participants received a detailed explanation regarding the study objectives, procedures, potential risks, and benefits. **Written informed consent** was also obtained before participation, ensuring compliance with the **Declaration of Helsinki on Human Research Ethics.**

### **Inclusion Criteria**

* Male commercial drivers aged **25–55 years.**
* Minimum of **one year of professional driving experience.**
* Regular consumption of alcohol and/or NSAIDs (self-reported).

### **Exclusion Criteria**

* Diagnosed cases of **diabetes mellitus, hypertension, chronic kidney disease, or other metabolic syndromes.**
* History of **renal disorders or recent hospitalization** due to kidney-related conditions.
* Recent use of nephrotoxic medications other than NSAIDs.

## **Data Collection**

### **Questionnaire Administration**

A **structured questionnaire** was administered to collect:

1. **Sociodemographic data** (age, education level, driving experience).
2. **Medical history** (pre-existing conditions, medication use).
3. **Lifestyle factors** (alcohol and NSAID consumption patterns, smoking, dietary habits).
4. **Occupational exposure** (average daily driving hours, stress levels).

To address literacy barriers, the questionnaire was available in **English, Hausa, Yoruba, and Pidgin English,** and an interviewer-assisted approach was used.

### **Blood Sample Collection**

**5 mL of venous blood** was drawn from each participant using aseptic techniques and aliquoted into **Fluoride oxalate tubes** for glucose analysis**, Lithium heparin tubes** for creatinine, urea, and electrolyte analysis,and **Plain tubes** for serum-based tests

### **Sample Processing**

Blood samples were **centrifuged at 2500 rpm for 10 minutes** to separate plasma/serum and Plasma was stored at **-20°C** until biochemical analysis(14).

## **Biochemical Analysis**

### **Glucose Determination (Glucose Oxidase-Peroxidase Method, Trinder, 1969)**(15)

Glucose levels will be determined enzymatically using the glucose oxidase-peroxidase (GOD-POD) method.

#### **Procedure**

**10 L of plasma sample was pipetted** into a test tube, and **1,000 µL of glucose reagent** was added. A standard curve was prepared using **10 µL of glucose standard + and 1,000 µL of reagent. 1,000 µL of reagent was used as a blank** and incubated in the tubes at **37°C for 10 minutes**. Absorbance was measured at **560 nm** using a spectrophotometer.

#### ****Calculation****

Concentration of test = Optical density of test X concentration of the standard

 Optical density of standard

#### ****Principle****

Creatinine in an **alkaline medium** reacts with **picric acid** to form an orange-red complex. The intensity of the color is proportional to creatinine concentration.

#### **Reagent Composition:Picric acid** 35 mmol/L, **Sodium hydroxide**: 0.32 mmol/L **Creatinine standard**: 177 μmol/L

#### **Procedure**

**Equal volumes of picric acid and sodium hydroxide were mixed** to prepare the working reagent; **500 µL of reagent** was added to **100 µL of the plasma sample.** Incubated at **37°C for 10 minutes**. Absorbance measured at **492 nm** using a spectrophotometer.

### **Urea Estimation (Urease-Berthelot Reaction, Weatherburn, 1967)**

#### ****Principle****

Urease hydrolyzes **urea into ammonia** and carbon dioxide. The ammonia reacts with hypochlorite and salicylate to form a **green-colored complex**.

#### ****Procedure****

1. **of urea, reagent** was added to **100 µL of the plasma sample.** Incubate at **37°C for 5 minutes.** Absorbance was measured at **540 nm** using a spectrophotometer.

### **Electrolyte Analysis (Ion-Selective Electrode, ISE Method)**

Plasma **potassium levels** were determined using an **ion-selective electrode (ISE) analyzer**, which provides **rapid and accurate electrolyte measurements.**

## **Statistical Analysis**

Data was analyzed using **SPSS version 25.0**. Descriptive statistics were presented as **mean ± standard deviation (SD)** for continuous variables and **percentages for categorical variables.** Group differences were assessed using **independent t-tests** and **chi-square tests,** while **multiple regression analysis** was performed to determine associations between substance use and renal function markers. A **p-value < 0.05** will be considered statistically significant.

## **RESULTS AND FINDINGS**

Table 1 and Fig. 1 present the demographic distribution of participants. The majority of participants were male, with similar age distributions between the test and control groups.

#### **Table 1: Demographic Characteristics of Participants**

| Variable | Test Group (n=33) | Control Group (n=33) | p-value |
| --- | --- | --- | --- |
| **Gender** |  |  |  |
| Male | 90.9% | 87.9% | - |
| Female | 9.1% | 12.1% | - |
| **Age Group** |  |  |  |
| 21-30 years | 60.6% | 57.6% | 0.448 |
| 31-40 years | 30.3% | 36.4% |  |
| 41-50 years | 3.0% | 6.1% |  |
| 51-60 years | 6.1% | - |  |

**Key Findings:** most of the participants were aged **21-30 years**, with no significant difference observed in age distribution between groups.

### **Fig 1: Demographic Characteristics**

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#### ****Prevalence of Hypertension and Glucose Abnormalities****

Table 2 and Figure 2 compare the prevalence of **hypertension and glucose abnormalities** between groups.

#### ****T**able 2: Hypertension and Glucose Levels Between Test and Control Groups**

| Condition | Test Group (%) | Control Group (%) | p-value |
| --- | --- | --- | --- |
| **Diastolic Hypertension**  | 36.4% | 15.2% | 0.015\* |
| **Systolic Hypertension** | 48.5% | 12.1% | 0.002\*\* |
| **High Blood Glucose** | 39.4% | 30.3% | 0.509 |

**Key Findings: The test group was reported to be more hypertensive than the control group. (p<0.05)**. The **blood glucose levels of the test group were also high but** considered statistically significant (**p = 0.509).**

Figure2 

#### ****Electrolyte and Renal Function Markers****

Table 3 and Figure 3 shows the distribution of renal function and electrolyte markers.

#### ****Table 3: Comparison of Renal Function and Electrolytes****

| Test | Category | Test Group (%) | Control Group (%) | p-value |
| --- | --- | --- | --- | --- |
| **Potassium Level** | Low | 42.4% | 12.1% | 0.001\*\* |
|  | Normal | 57.6% | 87.9% | - |
| **Urea Level** | High | 15.2% | 0% | 0.000\*\* |
|  | Low | 6.1% | 0% | - |
|  | Normal | 78.8% | 100% | - |
| **Creatinine Level** | High | 12.1% | 0% | 0.152 |
|  | Low | 3.0% | 0% | - |
|  | Normal | 84.8% | 100% | - |

**Key Findings:** The **potassium and urea levels** **(p<0.05)** were reported to be **significantly different,** suggesting a possible **renal impairment**. No significant difference was seen in **Creatinine levels** between groups **(p=0.152).**

**Figure 3**



#### ****Association Between Renal Function and Hypertension****

Table 4 and Figure 4 summarize the relationship between **renal markers (urea, creatinine) and hypertension**.

#### **Table 4: Association Between Renal Function and Hypertension**

| Test | Hypertensive (%) | Normotensive (%) | p-value |
| --- | --- | --- | --- |
| **Urea Level** | 36.4% | 63.6% | 0.674 |
| **Creatinine Level** | 36.4% | 63.6% | 0.638 |

**Key Findings: No significant relationship** was observed between **urea or creatinine levels and hypertension** (**p>0.05**). These findings proposed that **hypertension in this population may not be a result of renal function markers**.

**Figure4: 4**

### **Key Statistical Tests**

| Comparison | t-value | p-value | Interpretation |
| --- | --- | --- | --- |
| **Test vs Control Age** | 0.768 | 0.448 | Not significant |
| **Test vs Control Glucose** | 0.667 | 0.509 | Not significant |
| **Test vs Control Potassium** | -3.669 | 0.001\*\* | Significant |
| **Test vs Control Urea** | 4.019 | 0.000\*\* | Significant |
| **Test vs Control Creatinine** | 1.468 | 0.152 | Not significant |

(p < 0.05 indicates a significant difference)

**Urea and potassium levels were significantly different between the test and control groups,** indicating possible renal function impairment among alcohol-consuming drivers. Other biochemical parameters showed no significant differences.

## **Summary of Findings**

* **A higher prevalence of hypertension was seen** among commercial drivers than the control group, though not statistically significant.
* **Higher levels of Glucose i**n the test group but not significantly different from controls.
* Significant differences in **Renal function markers:** urea and potassium levels suggest potential renal dysfunction among alcohol-consuming drivers.
* **Statistical Significance** differences in urea and potassium levels (p < 0.05), while creatinine levels were not.

## **DISCUSSION**

The connection between alcohol use disorder (AUD) and kidney dysfunction is multifaceted and has continued to be a mystery that needs to be tackled. Previous epidemiological studies have suggested a potential relationship between chronic alcohol consumption and renal impairment, but there is no conclusive experimental evidence(16). The method by which alcohol can enhance kidney injury is still understudied but believed to involve oxidative stress, inflammation, and electrolyte imbalances(17). In this present study, we specifically aimed to examine the prevalence of renal dysfunction and hypertension among commercial bus drivers who consume alcohol and nonsteroidal anti-inflammatory drugs (NSAIDs), a population more vulnerable to occupational stress, lifestyle, and dietary habits(18).

###  **Renal Function and Alcohol Consumption**

Serum creatinine is a universally explored biomarker for assessing renal function, still, the way it is being interpreted may be impacted by some factors like muscle mass, how hydrated a person is, and dietary intake(19). Our study reported that 12.1% of participants showed highly elevated serum creatinine levels. Regardless, the mean serum creatinine concentration among alcohol consumers was not significantly different from that of nonconsumers (p>0.05). This finding is in alignment with Demnitiz et al., 2020, who reported that no significant relationship was seen between alcohol consumption and creatinine levels among commercial bus drivers(20). Although this showed that modesty in alcohol consumption may probably not affect renal function, it still does not eliminate subclinical kidney injury or long-term deterioration, which creatinine may not have captured(21). Glomerular filtration rate (GFR) is still the gold standard for assessing renal function, we recommend that future studies consider direct GFR measurements to better explicate the effect of alcohol consumption on renal health(22).

###  **Potassium Dysregulation and Alcohol Use**

The kidneys are a very important organ in maintaining the body's electrolyte balance. We found that **42.4% of the participants suffered from hypokalemia,** which is a statistically significant deviation from the control group (**p<0.001**). This aligns with previous research that reported that alcohol consumption may lead to the depletion of potassium. The mechanisms behind this remain a mystery but may be due to increased renal excretion, hormonal imbalances, or altered fluid regulation(23).

Potassium depletion has clinical relevance as it can lead to **hyponatremia, muscle weakness, arrhythmias, and impaired neuromuscular function.** Additionally, previous studies have suggested that a shortage of potassium in the body may **stimulate antidiuretic hormone (ADH) activity,** leading to fluid retention and potentially worsening sodium imbalance. This report necessitates the need for **electrolyte monitoring in chronic alcohol consumers,** particularly among individuals exposed to additional risk factors such as **dehydration, NSAID use, and poor dietary intake.**

### **Prevalence and Risk Factors for Hypertension**

We also reported a prevalence of 15% for a hypertensive population, which is lower than the elevated number reported by Lakshman et al., 2014 in South India (18), but could be compared to the **20.5% prevalence reported by Adesola et al., 2014 among commercial drivers in Nigeria** (24). This suggests regional variability in hypertension risk, likely influenced by lifestyle, dietary patterns, and healthcare access.

Driving encourages a sedentary lifestyle, stress, and poor diet, all of which have been established to be risk factors for hypertension. The connection between **body mass index (BMI) and hypertension** seen in this study aligns with previous reports on obesity being a key driver of elevated blood pressure. Additionally, our results showed that an increase in **age and BMI (≥25 kg/m²) are notable indications of hypertension,** further emphasizing the role of metabolic factors in cardiovascular risk among commercial drivers(25).

Interestingly, we saw **no significant link between hypertension and urea, creatinine, or potassium levels (p>0.05).** This showed that even though renal dysfunction and hypertension may coexist, **hypertension as seen in this population was more likely as a result of lifestyle rather than direct renal impairment.** This is similar to previous studies that suggested hypertension and kidney disease exist independently, especially in early disease stages(26).

###  **Clinical and Public Health Implications**

The outcome of this study has broader **clinical and occupational health implications. Owing to** the **high prevalence of electrolyte imbalances and hypertension,** strategized measures such as **routine health checkups, dietary modifications, and lifestyle changes** should be advocated for commercial drivers(27). Blood pressure and renal monitoring of these drivers should be regularly monitored to ensure early detection and management of hypertension and electrolyte imbalance. **Public health education** on the potential risks associated with excessive alcohol and NSAID consumption, especially their potential impact on renal and cardiovascular health(28). **Nutritional counseling, exercise, and stress management** should be encouraged to mitigate the long-term health risks associated with commercial driving.

### **Study Limitations and Future Research Directions**

Regardless of how strong the study seems to be, there are several limitations we can acknowledge

1. **Lack of direct GFR measurement:** Serum creatinine is an indirect marker of renal function, and **cystatin C or estimated GFR (eGFR)** could be employed to provide a more accurate assessment.
2. **Potential underreporting of alcohol and NSAID use:** Bias is inevitable in self-reported data. Future studies should incorporate **biomarkers of alcohol consumption (e.g., blood ethanol levels, carbohydrate-deficient transferrin [CDT])** for a more objective assessment void of bias
3. **Cross-sectional design: our study captured the** associations but did not establish causality. Longitudinal studies are employed to determine this population's long-term renal and cardiovascular outcomes.
4. **Limited control for confounders:** although we controlled for BMI and age, other potential confounders such as **smoking, dietary sodium intake, genetic predisposition, and physical activity levels** were not fully assessed.

Future research should adopt a diverse approach **that** combines **biochemical, genetic, and epidemiological data** to holistically understand the interrelationship between alcohol, renal dysfunction, and hypertension in high-risk occupational groups.

### **Conclusion**

This present study underscores the **prevalence of renal dysfunction, electrolyte imbalances, and hypertension** among commercial bus drivers who consume alcohol and NSAIDs. Although no straightforward relationship was found between alcohol consumption and creatinine levels, the **high rate of hypokalemia and hypertension suggests that alcohol use may cause systemic metabolic challenges.** The findings present the need **for occupational health programs, routine medical assessments, and lifestyle interventions** to prevent long-term renal and cardiovascular complications in this high-risk population. **Future research should employ a broad approach** incorporating **biochemical, clinical, and epidemiological analyses** to better understand alcohol-related renal risks. By addressing these gaps, we can **improve health policies in the workplace, mitigate the risk of kidney disease and hypertension, and generally improve the health of commercial bus drivers.**

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