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*Full Length Research Paper*

## **Effect of Resin Extract from *Commiphora swynnertonii* (Burseraceae) on Biochemical Parameters In Rats**

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### **ABSTRACT**

The biological activities of various *Commiphora* plants (Burseraceae) have progressively been studied over a decade. Recent studies have established the ethnomedicinal potency of various *Commiphora* species in Africa and Asia. In this study, the effect of *Commiphora swynnertonii* resin on biochemical parameters in rats was investigated. Sixty rats were randomly assigned into five groups (n= 12) (G1 – G5). Group 1 (G1)- the control, received oral distilled water daily throughout the experiment. Group 2 (G2) received oral resin at a dose of 50 mg/kg body weight daily. Group 3 (G3) received oral resin at a dose of 100 mg/kg body weight while group 4 (G4) and group 5 (G5) received oral resin at doses 150 and 200 mg/kg body weight respectively daily throughout the experiment. Blood samples were taken after five days of treatment and assessed for plasma levels of glucose, total protein, albumin, triglycerides, total cholesterol, high density lipoprotein cholesterol (HDL-c), low density lipoprotein cholesterol (LDL-c), total bilirubin, liver enzymes; alanine aminotransferase (ALT), aspartate aminotransferase (AST) and alkaline phosphatase (ALP). Results revealed that oral administration of *Commiphora swynnertonii* resin at the dose range of 100 to 200 significantly decreased serum glucose, triglycerides, total cholesterol and LDL-c while increasing HDL-c. The resin also significantly elevated protein and albumin levels but had no significant effect on bilirubin and all the liver enzymes in the rats. These findings are suggestive of antidiabetic and hypolipidemic potential of *C. swynnertonii* resin and indicate that the extract could possess efficacy against cardiovascular disease and may improve liver synthetic function.

Key words: *Commiphora swynnertonii*, resin, biochemical parameters, rats

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### **INTRODUCTION**

The population medicinal plant comprises about 250,000 species, making up about 70% of the world's plants, accounting for about 50% of the drugs commonly derived from tropical plants (Akerle, 1992; Mamedov, 2012). The World Health Organization (WHO) (1996) recognizes medicinal plants as the primary source of health care to more than 80% of developing countries. The contemporary struggle for drug discovery from medicinal plants involves an interdisciplinary approach, combining botany, pharmacology, ethnology, and anthropology (Kayombo *et al.*, 2013) and the techniques include botanical, phytochemical, biological and molecular techniques, which in combination provide a clue towards tackling various pathological conditions such as

cancer, HIV/AIDS, malaria, Alzheimer's disease and many more (Balunas and Kinghorn, 2005). In Africa and Asia plants of in the genus *Commiphora* have been extensively used as medicinal plants (Kayombo *et al.*, 2013). *Commiphora swynnertonii* (*C. swynnertonii*), is among the plants that have been used by some Tanzanian tribes to treat various diseases in humans and animals (Bakari *et al.*, 2011, Mkangara *et al.*, 2014).

Different extracts from various *Commiphora* species have been broadly reported to possess, hypolipidemic, antihypertensive, antioxidant, liver-function boosting, antimicrobial, as well as anti-inflammatory properties (Shen *et al.*, 2012; Hassan *et al.*, 2013; Bakari *et al.*, 2015). *Commiphora mukul* (*C. mukul*) is a well studied species and is reported by many sources to be a potent medicinal plant

(Panda *et al.*, 1999; Sharma *et al.*, 2009; Hassan *et al.*, 2013). Extracts from *C. mukul* have demonstrated hepatoprotective and antioxidant activities by improving liver enzyme activities (ALT, AST and ALP) and the morphology of hepatocellular major organelles on carbon tetrachloride (CCl<sub>4</sub>) - induced liver injury in rats (Hassan *et al.*, 2013). In two studies, oral administration resin from *C. mukul* to mice decreased hepatic lipid peroxidation meanwhile elevating triiodothyronine (T3) and thyroxin (T4) levels, suggesting that that plant is effective against hypothyroidism (Panda *et al.*, 1999; Panda *et al.*, 2005).

The efficacy of resins obtained from *C. mukul* and *C. molmol* against hyperlipidemia and schistosomiasis has already been substantiated by pharmacological results and therefore validated for the treatment of the diseases in Egypt and India (Shen *et al.*, 2012). Guggulsterone, a bioactive steroid compound isolated from *C. mukul* resin, has exhibited both hypoglycemic and hypolipidemic effects against high fat diet-induced diabetes mellitus in rats, suggesting its potential in treating type 2 diabetes mellitus (Sharma *et al.*, 2009).

Different morphological parts of *C. swynnertonii* have been reported to possess several biological effects including antimicrobial activities *in vitro* and in chickens (Bakari *et al.*, 2013; Mkangara *et al.*, 2014; Bakari *et al.*, 2015). However to best of our knowledge no study has so far reported the effect of *C. swynnertonii* resin on biochemical parameters in rats. The current study therefore aimed at determining the effect of *C. swynnertonii* resin on biochemical parameters for the sake of extrapolating the knowledge on the biological activity of the plant from antimicrobial to other parameters of physiological importance in rats.

## MATERIALS AND METHODS

**Source of study material, study area and preparation of the study material:** Plant material was obtained from Arumeru District in Arusha Region (Northeastern Tanzania) and transported to Marian University College where preparation was done in the Biology laboratory according to Bakari *et al.* (2011). The solution was then stored in clean bottles at 4°C until experimentation.

**Experimental design:** Sixty male rats were randomly assigned into five groups (n= 12). The first group (G1) was a control, receiving distilled water daily throughout the experiment. Group 2 (G2) received oral resin dally 50 mg/kg body weight. Group 3 (G3) received oral resin at a dose of 100 mg/kg body weight while group 4 (G4) and group 5 (G5) received oral resin at doses 150 and 200 mg/kg body weight respectively daily throughout the experiment. Blood samples were taken every after five days of treatment and assessed for plasma glucose, total protein, albumin, triglycerides, total cholesterol, high density lipoprotein cholesterol (HDL-c), low density lipoprotein cholesterol (LDL-c), total bilirubin, liver enzymes; alanine aminotransferase (ALT), aspartate aminotransferase (AST) and alkaline phosphatase (ALP).

**Blood sample collection, preparation biochemical analyses:** Blood samples were collected from lateral tail veins using a 5-mL syringe. The samples were then stored in heparin sterile vacutainer tubes. The blood samples were centrifuged at 1300 rpm for 5 minutes to separate plasma. The plasma samples were stored in a refrigerator (4 °C) until they were used for analysis of various biochemical parameters. All laboratory analyses were carried out using standard method kits (Erba® Diagnostics, Mannheim, Germany).

## Data analysis

Statistical analysis of data was accomplished by means of the SAS for Microsoft windows statistical software package (2007). One way analysis of variance (ANOVA) was used to compare parameters among the groups whereas correlation analysis was also employed to follow up the trend of lipid variation and weight change over time.

## RESULTS

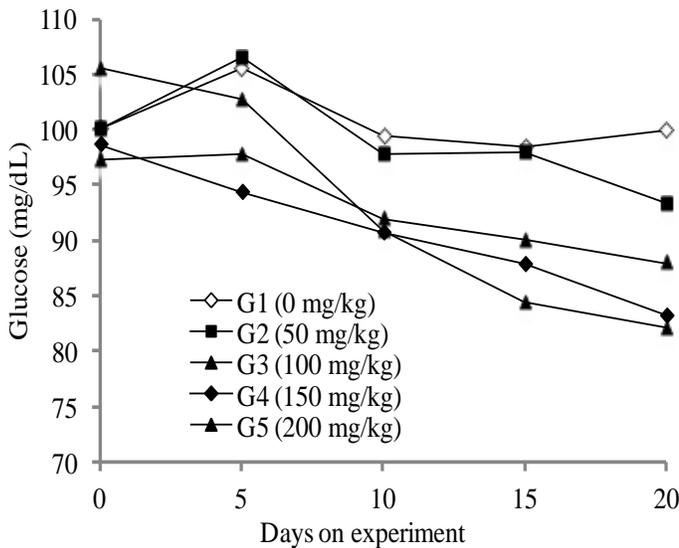
**Effect of resin on plasma glucose:** No significant change in the levels of plasma glucose among rats in the control group (0 mg resin/kg bw) and in G2 (50 mg resin/kg bw). The rats that were treated with 100 mg, 150 mg and 200 mg/kg bw (G3, G4, and G5 respectively) showed a significant decrease ( $p < 0.05$ ) in plasma glucose from day 10 to day 20 of the experiment (Fig 1), with the highest dose inducing the greatest decrease on day 20 (indicating a dose and time dependent manner;  $R^2 = 0.95$ ,  $p = 0.003$ ).

### Effects of *C. swynnertonii* resin on plasma lipids

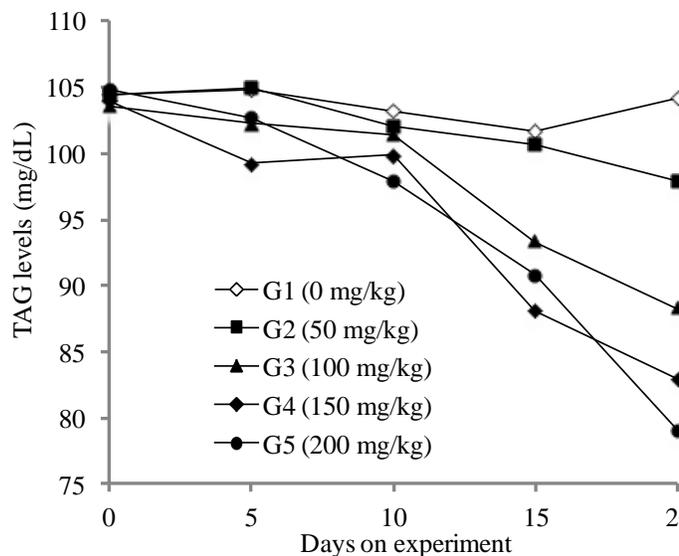
**Triacylglycerol (TAG) levels:** There was no significant change ( $p > 0.05$ ) in G1 (control group) and the animals treated with 50 mg resin /kg bw (G2) but those treated with 100 mg, 150 mg and 200 mg resin/kg bw (G3, G4 and G5 respectively) showed significantly ( $p < 0.05$ ) lower plasma TAG levels from day 10 towards the end of the experiment.

Figure 2 above demonstrates that the effect of resin on TAG levels was dose and time dependent ( $R^2 = 0.89$ ). For example, on day 15 and on day 20 the mean TAG levels of rats in G4 (150 mg resin/kg bw) were 88.1 and 83.0 mg/dL respectively while those of G5 (200 mg resin/kg bw) were 90.8 and 79.1 mg/dL respectively.

**Effect of resin from *C. swynnertonii* on plasma total cholesterol levels:** There was no significant change in the levels of plasma cholesterol among animals in the control group. There was a dose and time dependent decrease in plasma cholesterol following oral administration of *C. swynnertonii* resin ( $R^2 = 0.97$ ). Resin at the dose 50 mg resin/kg bw did not significantly alter the levels throughout the experiment whereas doses 100, 150 and 200 mg resin/kg bw resulted in significant decrease ( $p < 0.05$ ) in plasma cholesterol levels of rats in G3, G4 and G5 respectively (Fig. 3).



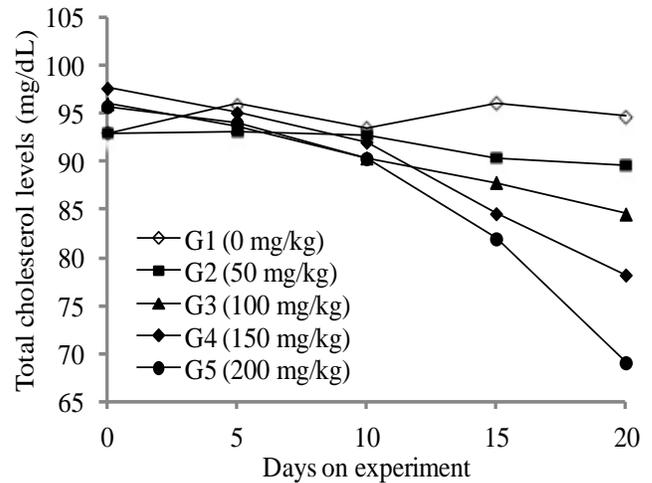
**Fig. 1:** Plasma glucose levels following oral administration of *C. swynnertonii* resin



**Fig 2:** Plasma levels of triacylglycerols following administration of *C. swynnertonii* resin

**Effect of resin from *C. swynnertonii* on lipoprotein cholesterol forms:**

As shown in Table 1, rats in the control group did not show any significant change in levels of all the forms of cholesterol. Similarly, no significant change in all the three cholesterol forms was observed in the groups treated with doses of 50 and 100 mg/kg resin/kg bw throughout the experiment while the doses 150 and 200 mg resin/kg bw were associated with significant decline in VLDL-c and LDL-c but elevation in HDL-c plasma levels from day 15 towards the end of the experiment.



**Fig. 3:** Plasma total cholesterol profile following administration of *C. swynnertonii* resin

**Effects of *C. swynnertonii* resin extract on liver function biomarkers**

**Plasma total protein and albumin levels:**

Rats in the two groups G1 (control group) G2, (50 mg resin/kg bw) and G5 (200 mg resin/kg bw) had no significant change in the levels of plasma total protein and albumin throughout the experiment (Table 2). Following oral administration of *C. swynnertonii* resin, plasma total protein and albumin increased significantly ( $p < 0.05$ ) in G3 and G4 (100 and 150 mg resin/kg bw respectively) from day 15 towards day 20.

**Effects of oral administration of *C. swynnertonii* resin on bilirubin, ALT, AST and ALP activities in the experimental rats:**

No significant change in the levels of bilirubin was observed in all the groups. Similarly, (Table 3), oral administration of resin at all the four doses did not significantly alter the activities of all the three enzymes despite minor fluctuations towards the end of the experiment.

**Other observations:**

Body weight showed a decreasing trend with decrease in cholesterol. Animals in all the resin treated groups were observed to lose weight over time. For example, (Fig. 4 and Fig. 5) there was a positive correlation between cholesterol level decline and weight loss in G4 and G5 animals (correlation coefficient = 0.98).

Figures 4 and 5 indicate that there was a negative correlation between increase in the resin dose and plasma total cholesterol (coefficient of correlation = -0.95) and between resin dose and body weight loss (coefficient of correlation = -0.96). For example on day 20, animals treated with 150 mg resin/kg bw (Fig. 4) had a mean weight of  $170.4 \pm 8.5$  g whereas those (Fig. 5) treated with the highest resin dose (200 mg resin/kg bw) had the lowest mean weight ( $163.6 \pm 8.1$  g).

**Table 2:**

Levels of differential cholesterol forms over experiment time (means ± standard errors of the means)

Parameter	Group/ dose	Treatment	Days on experiment				
			0	5	10	15	20
HDL-c (mg/dL)	G1	0 mg resin per kg b.w	34.0 ± 1.0	36.6 ± 2.4	34.6 ± 0.7	30.9 ± 2.8	34.3 ± 0.6
	G2	50 mg resin per kg b.w	34.0 ± 1.0	34.3 ± 1.0	34.0 ± 1.4	34.7 ± 1.4	35.2 ± 1.5
	G3	100 mg resin per kg b.w	33.5 ± 1.0	33.7 ± 1.0	35.1 ± 1.0	36.0 ± 0.8	37.8 ± 0.7
	G4	150 mg resin per kg b.w	32.7 ± 0.8	32.0 ± 1.2	33.5 ± 1.4	38.4 ± 0.9*	38.1 ± 1.0*
	G5	200 mg resin per kg b.w	34.8 ± 1.1	36.4 ± 2.5	36.4 ± 2.5	38.2 ± 1.3*	38.6 ± 1.2*
LDL-c (mg/dL)	G1	0 mg resin per kg b.w	38.1 ± 1.9	38.4 ± 2.4	38.3 ± 1.5	44.8 ± 2.6	39.5 ± 1.4
	G2	50 mg resin per kg b.w	38.1 ± 2.0	37.9 ± 1.9	38.4 ± 1.9	35.4 ± 2.4	34.7 ± 2.3
	G3	100 mg resin per kg b.w	42.0 ± 1.9	39.5 ± 2.1	36.7 ± 2.3	29.1 ± 2.2	29.1 ± 1.9
	G4	150 mg resin per kg b.w	44.2 ± 1.4	38.4 ± 1.9	28.3 ± 1.9*	28.3 ± 1.4*	23.5 ± 1.9*
	G5	200 mg resin per kg b.w	39.9 ± 1.7	37.1 ± 2.7	34.3 ± 2.2	25.3 ± 2.0*	14.0 ± 2.*1
VLDL-c (mg/dL)	G1	0 mg resin per kg b.w	20.9 ± 0.3	21.0 ± 0.4	20.6 ± 0.3	20.5 ± 0.3	20.8 ± 0.3
	G2	50 mg resin per kg b.w	20.9 ± 0.3	21.0 ± 0.2	20.5 ± 0.2	20.3 ± 0.3	19.8 ± 0.3
	G3	100 mg resin per kg b.w	20.6 ± 0.3	20.4 ± 0.3	18.6 ± 0.7	19.0 ± 0.6	17.7 ± 0.8
	G4	150 mg resin per kg b.w	20.8 ± 0.3	19.9 ± 0.4	20.1 ± 0.3	17.9 ± 0.5	16.7 ± 0.6*
	G5	200 mg resin per kg b.w	21.0 ± 0.4	20.6 ± 0.4	19.7 ± 0.4	18.5 ± 0.4	16.5 ± 0.6*

\*p < 0.05 vs. control

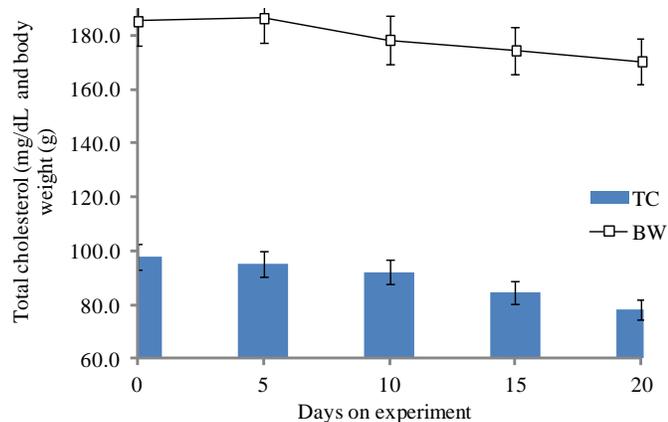
**Table 3:** Total plasma protein and albumin levels following oral administration of resin (means ± standard errors of the means).

Parameter	Group (dose)	Days on experiment				
		0	5	10	15	20
Total protein (g/dL)	G1 (0 mg/kg)	5.6 ± 0.2	5.7 ± 0.4	5.9 ± 0.1	5.9 ± 0.1	6.0 ± 0.1
	G2 (50 mg/kg)	5.4 ± 0.1	5.9 ± 0.1	6.0 ± 0.1	6.2 ± 0.1	6.2 ± 0.1
	G3 (100 mg/kg)	5.5 ± 0.1	5.6 ± 0.1	6.3 ± 0.1	6.4 ± 0.1*	6.7 ± 0.1*
	G4 (150 mg/kg)	5.5 ± 0.2	5.5 ± 0.2	5.9 ± 0.2	6.4 ± 0.1*	6.8 ± 0.1*
	G5 (200 mg/kg)	5.4 ± 0.2	5.7 ± 0.2	6.1 ± 0.1	5.7 ± 0.2	5.5 ± 0.1
Albumin (g/dL)	G1 (0 mg/kg)	3.5 ± 0.1	3.6 ± 0.1	3.6 ± 0.1	3.7 ± 0.1	3.5 ± 0.1
	G2 (50 mg/kg)	3.5 ± 0.1	3.5 ± 0.1	3.6 ± 0.1	3.5 ± 0.1	3.8 ± 0.1
	G3 (100 mg/kg)	3.5 ± 0.1	3.6 ± 0.1	3.8 ± 0.1	3.9 ± 0.1*	4.0 ± 0.1*
	G4 (150 mg/kg)	3.7 ± 0.1	3.7 ± 0.2	3.7 ± 0.2	4.0* ± 0.1	4.1 ± 0.1*
	G5 (200 mg/kg)	3.6 ± 0.1	3.7 ± 0.1	3.7 ± 0.2	3.5 ± 0.2	3.3 ± 0.1

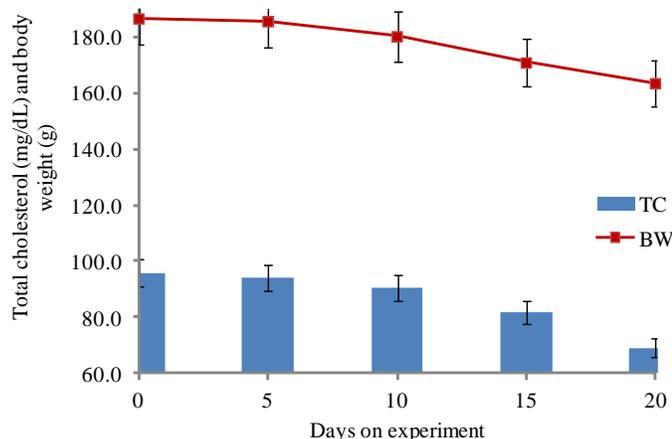
\*p < 0.05 vs. control

**Table 4:** Profile of liver function markers over experimentation (means ± standard errors of the means)

Parameter	Group	Days on experiment					
		0	5	10	15	20	
BIL (g/dL)	G1	G1 (0 mg/kg)	3.7 ± 0.2	3.4 ± 0.1	3.6 ± 0.1	3.6 ± 0.1	3.7 ± 0.1
	G2	G2 (50 mg/kg)	3.7 ± 0.2	3.7 ± 0.1	3.6 ± 0.1	3.5 ± 0.1	3.6 ± 0.1
	G3	G3 (100 mg/kg)	3.6 ± 0.1	3.6 ± 0.1	3.5 ± 0.1	3.5 ± 0.1	3.5 ± 0.1
	G4	G4 (150 mg/kg)	3.4 ± 0.1	3.6 ± 0.1	3.4 ± 0.1	3.3 ± 0.1	3.4 ± 0.1
	G5	G5 (200 mg/kg)	3.6 ± 0.1	3.4 ± 0.1	3.4 ± 0.1	3.5 ± 0.1	3.8 ± 0.1
ALT (µ/L)	G1	G1 (0 mg/kg)	23.9 ± 0.9	25.3 ± 1.3	24.7 ± 1.1	26.9 ± 1.8	25.0 ± 1.2
	G2	G2 (50 mg/kg)	24.7 ± 1.9	22.7 ± 1.9	22.9 ± 2.1	24.1 ± 1.3	24.1 ± 1.2
	G3	G3 (100 mg/kg)	22.6 ± 0.9	23.7 ± 1.0	24.0 ± 0.8	23.6 ± 1.0	22.8 ± 1.1
	G4	G4 (150 mg/kg)	25.1 ± 1.2	26.1 ± 1.3	24.9 ± 1.4	23.9 ± 1.0	23.7 ± 1.0
	G5	G5 (200 mg/kg)	25.3 ± 1.3	24.8 ± 1.5	24.4 ± 1.3	26.5 ± 1.3	23.6 ± 0.8
AST (µ/L)	G1	G1 (0 mg/kg)	48.4 ± 1.4	49.2 ± 1.5	48.4 ± 1.5	48.9 ± 1.4	47.5 ± 1.4
	G2	G2 (50 mg/kg)	48.4 ± 1.5	48.0 ± 1.6	47.4 ± 1.4	49.5 ± 1.3	48.8 ± 1.4
	G3	G3 (100 mg/kg)	47.0 ± 1.5	47.5 ± 1.7	45.5 ± 1.2	44.5 ± 1.5	43.7 ± 1.2
	G4	G4 (150 mg/kg)	46.7 ± 2.0	46.0 ± 1.9	45.1 ± 1.4	44.3 ± 1.5	42.7 ± 1.3
	G5	G5 (200 mg/kg)	49.0 ± 1.5	46.4 ± 1.5	47.6 ± 1.0	47.4 ± 1.3	52.6 ± 1.5
ALP (µ/L)	G1	G1 (0 mg/kg)	68.3 ± 2.1	71.5 ± 2.0	68.4 ± 2.2	68.3 ± 2.3	68.6 ± 2.1
	G2	G2 (50 mg/kg)	65.9 ± 1.9	66.4 ± 1.9	65.7 ± 1.4	68.6 ± 1.2	65.3 ± 1.2
	G3	G3 (100 mg/kg)	70.0 ± 1.6	68.2 ± 1.5	70.0 ± 1.5	66.4 ± 1.2	65.2 ± 1.2
	G4	G4 (150 mg/kg)	68.9 ± 1.6	70.0 ± 2.0	68.0 ± 2.2	68.7 ± 2.0	65.4 ± 1.9
	G5	G5 (200 mg/kg)	71.5 ± 2.0	67.9 ± 2.7	71.4 ± 2.3	74.1 ± 2.4	76.2 ± 2.9



**Fig. 4:** Correlation between plasma total cholesterol and body weight variations (mean  $\pm$ percentage error) among (150 mg resin/kg bw) G4 rat



**Fig 5:** Correlation between plasma total cholesterol and body weight variations (mean  $\pm$ percentage error) among (200 mg resin/kg bw) G5 rats

## DISCUSSION

*Commiphora swynnertonii* resin has demonstrated a range of effects on various biochemical parameters in rats. Resin extract from *C. swynnertonii* has revealed a hypoglycemic effect in the rats. Figure 2 demonstrates that oral administration of doses above 50 mg resin/kg bw was effective against plasma glucose levels in the rats and the effect was both dose and time dependent. For example on day 15 the percentage decrease in glucose was 10.8% in group 4 rats (rats that were treated with 150 mg resin/kg bw) and 14.3% in group 5 rats (those treated with 200 mg resin/kg bw). On day 20, the animals in group 4 exhibited a 16.8% decrease in glucose against 17.5% exhibited by those in group 5. These hypoglycemic findings have shown concurrence with several studies done using various extracts from different *Commiphora* species (Goji *et al.*, 2009; Al-Amoudi, 2010; Ramesh and Saralalukumari, 2012). In a study by Goji *et al.*

(2009), ethanolic extracts from *Commiphora africana* exhibited efficacy against glucose levels in normoglycemic rats. In another study by Al-Amoudi (2010), resin extract form *Commiphora myrrha* was found to decrease serum glucose levels by 37.24% in alloxan-induced diabetic rats. Furthermore, *Commiphora mukul* has been demonstrated to have hypoglycemic effects in fructose-induced hyperglycemic Wistar rats (Ramesh and Saralalukumari, 2012).

The hypoglycemic effect of *C. swynnertonii* resin could be attributed several phytochemicals such as flavonoids (Lukacinova., *et al.* 2008; Rauter, 2010; Brahmachari, 2011; Mahmoud, 2013) commonly reported from other *Commiphora* species including *Commiphora myrrha* (Helal, 2005) and *Commiphora africana* (Goji *et al.* 2009). The mechanism of reduction in plasma glucose by *Commiphora* resin may be mediated by insulin-like activity or activation of pancreatic beta-cells by the constituents (Goji *et al.* 2009).

The findings have shown that oral administration of *C. swynnertonii* is associated with decreased plasma TAGs, total cholesterol, LDL-c and VLDL-c and increased HDL-c. An interesting observation was that decline in the level of cholesterol (Fig. 5, Fig. 6) was associated with corresponding weight loss. In G4 (the rats were treated with 150 mg resin/kg bw), the percentage decreases in total cholesterol on day 15 and day 20 were 12.0% and 17.4% respectively, which corresponded to percentage body weight losses of 6.7% and 10.1 % respectively. The percentage effects were even higher in G5 (the rats that were treated with 200 mg resin/kg bw). On the same days 15 and 20, 14.7% and 27.1% cholesterol declines corresponded to 8.6 and 13.7% weight losses respectively. This observation indicates that the plant extract could manage cholesterol levels meanwhile controlling weight. Furthermore, the decrease in total cholesterol was associated with decrease in LDL-c (bad cholesterol) and increase in HDL-c (good cholesterol.), which suggests that the plant extract could be useful in managing fats and therefore lowering the risk of cardiovascular disease (Wang *et al.*, 2004).

The ability of *C. swynnertonii* to lower lipid levels is comparable to findings from other studies in rats and chickens (Adebayo *et al.*, 2006; Bakari *et al.*, 2015). The 27% hypocholesterolemic effect of *C. swynnertonii* resin is closely related to that of 24% leaf ethanolic extracts from *C. africana* (Adebayo *et al.*, 2006). The synergy between hypolipidemic and weight loss effects is the most interesting finding in this study, which has also been reported by Bakari *et al.* (2015) in chickens. Although the animals in this study were not challenged by any pathologic agent, these findings have demonstrated efficacy of the extract in improving good cholesterol while suppressing bad cholesterol, similar to those in which the rats were treated with diabetic agents (Al-Amoudi, 2010; Bellamkonda *et al.*, 2011; Ramesh *et al.*, 2012), and this calls for a further study to test the effect of the extract in the challenged animal models. The lipoprotein modulation supports the view that the resin could act by inhibiting lipid synthesis and modulating lipid mobilization via anti-peroxidant activity (Chanderet *et al.*, 2003).

Studies have elucidated the phytochemistry of many *Commiphora* species, from which the antilipidemic and mechanism of action of guggulsterone from *C. mukul* has been well substantiated (Urizar *et al.*, 2002; Cui *et al.*, 2003; Deng, 2007; Yang *et al.* 2012). However the phytochemistry of *C. swynnertonii* is poorly known. Thus studies are needed to elucidate the phytochemical ingredients in the resin as well as their mechanisms of action. Flavonoids, terpenoids and guggulipids may be some of the phytochemicals attributable to the desirable effects *C. swynnertonii* resin (Wang *et al.*, 2004; Siddiqui and Mazumder, 2012; Bhardwaj *et al.*, 2013).

The present work has shown the potency of *C. swynnertonii* resin in elevating plasma protein and albumin in the rats. However, the findings have shown a dose margin. As shown on Table 3, the lowest dose (50 mg resin/kg) was associated with insignificant elevations of protein and albumin whereas the highest dose was associated with insignificant declines in the parameters. This observation attracts attention on the dose range of 100 - 150 mg resin/kg bw if the experiment is to be done in 20 days on rats using the resin extract. This dose range might have a boosting effect on the synthetic activity of the liver (as depicted by protein level elevation). In addition, the observation that all the doses had no significant effect on bilirubin and liver enzymes indicates that the doses were not effective on the parameters over the experimental period. Moreover, these findings could imply that the extract has little or no toxic effect to the liver when administered at the dose range of 50 – 200 mg resin/kg bw over 20 days. These findings are supported by findings from Bakari *et al.* (2015) who found insignificant slight elevations of AST and ALT activities with exception that their study observed steady levels of plasma protein and albumin towards the end of the experiment done in chickens. The explanation for this deviation is unclear although it could be attributable to several factors among which include anatomical and/or morphological differences between rats and birds, which would influence the pharmacodynamics and pharmacokinetics of the resin ingredients, and physiological differences between rats and chickens. Although the current study also concurs with that of Aliyu *et al.* (2007) in which oral administration of ethanolic leaf extracts from *C. africana* to albino rats did not significantly alter AST and ALP activities, the two differ in that the leaf extract at dose 100 mg/kg bw in Aliyu *et al.* (2007) decreased ALT activity towards day 10 but did not alter albumin levels. These discrepancies might possibly be caused by differences in the plant species and extract ingredients, a speculation which calls for further investigation.

In conclusion, resin from *Commiphora swynnertonii* possesses hypoglycemic and hypolipidemic effects in rats in addition to its ability to boost liver function when administered with the dose range of 100 – 200 mg resin/kg bw. These desirable properties are compelling evidence that the *C. swynnertonii* resin can be a good source of antidiabetic, cardioprotective, liver function boosting and possibly antioxidant agents.

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