THE EFFECT OF VERNONIA AMYGDALINA AND OCIMUM GRATISSIMUM ON ALLOXAN-INDUCED DIABETIC RATS

Adewoga TOS, Sebiomo A, Fagbemi FT

Department of Biological Sciences, Tai Solarin University of Education, Ijagun, Ijebu-Ode, Nigeria

Corresponding author: Sebiomo A Email: rev20032002@yahoo.com

ABSTRACT

Aim: Anti-diabetic evaluation of Vernonia amygdalina and Ocimum gratissimum on alloxan induced diabetic rats was investigated.

Methods: Thirty rats were divided into six groups of five per group. The rats were fed with aqueous and ethanolic plant extracts of the plants.

Results: On day 2 post induction, fasting blood glucose of all the animals across the groups increased significantly (P<0.05) compared to pre induction across all the six groups for both water extract and ethanolic extract treated groups. On day 8 post induction (ethanolic extract), O. gratissimum had the lowest fasting blood glucose of 81 (±10.02) mg/dl when compared to other test groups while the highest fasting blood glucose of 600 (±0.00) mg/dl was observed in alloxan only group. On day 8 post induction (water extract) the control group, glibenclamide group, O. gratissimum group, all recorded reduced fasting blood glucose level of 98 (±6.35) mg/dl, 98.33 (±7.27) mg/dl, 105.67 (±10.02) mg/dl, 305.67 (±68) mg/dl respectively compared to values obtained on day 6 post induction. A drastic reduction in body weight of animals in groups treated with V. amygdalina, combined extract, glibenclamide and alloxan only can be observed (for both ethanolic and water extract treated groups). Out of all the extracts used, the extract of O. gratissimum significantly reduced the fasting blood glucose and increased the body weight of the rats at the end of the experiment.

Conclusion: O. gratissimum can be used to reduce elevated blood glucose of diabetic animals, and it proved to be more potent than V. amygdalina.

Key words: Ocimum gratissimum, Vernonia amygdalina, Alloxan, Glibenclamide

INTRODUCTION

Diabetes is an age long, serious metabolic disorder with complications that result in significant morbidity and mortality. According to Chukwuma (2012), chronic hyperglycemia during diabetes has been shown to cause glycation of

body protein, which in turn leads to secondary complications that affect the eyes, kidneys, nerves and arteries. Diabetes mellitus is characterized by disordered metabolism and abnormally high blood glucose resulting from insufficient levels of insulin. It has gradually found its root in Africa, especially in Nigeria where westernized ways are imbibed (Asuquo et al., 2010). Type 1 diabetes is also called Insulin-Dependent Diabetes mellitus (IDDM). It was initially called juvenile-onset diabetes, because it often begins in childhood. It is an auto immune condition, caused by the body attacking its own pancreas with antibodies. In people with type 1 diabetes, the damaged pancreas does not make insulin. This type of diabetes may be caused by genetic predisposition. It could also be the result of faulty beta cells in the pancreas that normally produce insulin. A number of medical risks are associated with this type 1 diabetes. Many of them damage the tiny blood vessels in the eyes (called diabetic retinopathy), nerves (diabetic neuropathy), and kidneys (diabetic nephropathy). Even more serious is the increased risk of heart disease and stroke. The most common form of diabetes is type 2 diabetes, also known as non-insulin dependent Diabetes mellitus (NIDDM), accounting for 95% diabetes cases in adults. It is used to be called adult onset diabetes, but with the epidemic of obese and overweight kids, more teenagers are now with type 2 diabetes. Type 2 diabetes is often a milder form of diabetes than type 1. Nevertheless, type 2 cause diabetes can still maior health complications, particularly in the smallest blood vessels in the body that nourish the kidneys, nerves and eves. It also increases the risk of heart disease and stroke. With type 2 diabetes, the pancreas usually produces some insulin, but either the amount is not enough for the body needs or the body's cells are resistant to it. The number of people with diabetes is increasing due to population growth, aging, urbanization, and increasing prevalence of obesity and physical inactivity (Sarah et al., 2000). Vernonia amygdalina, a member of Asetraceae is a small shrub that grows in the tropical Africa. It is commonly called bitter leaf because of its bitter taste. The leaves may be consumed either as a vegetable (macerated leaves) in soups or aqueous extracts as tonics for treatment of various illnesses. Vernonia amygdalina has been found useful in the ethnotheraphy of asthma, malaria, measles, diarrhea, tuberculosis, abdominal pain and fever (Chukwuma, 2012). The results of the phytochemical screening showed that carbohydrates, saponins, alkaloids, tannins. proteins and steroids occurred in very higher concentration, while flavonoids and glycosides

occurred in high concentration. Bitter leaf is able to influence some drop in sugar level. Ocimum gratissimum is of the family Labiatae, and is found throughout the tropics, subtropics and its greatest variability occurs in tropical Africa and India. It is commonly used in cooking due to its minty aromatic flavor. Traditionally, it has been used for the treatment of headache, diarrhea, warts. worms and kidnev infections. Phytochemical evaluation of this plant has shown that it is rich in alkaloid, tannin, phytates, flavonoids, and oligosaccharides. It has tolerable cyanogenic content (Efiri, 2012). Vernonia amygdalina and Ocimum gratissimum are among the traditionally used herbal plants for the treatment of different ailments (Asuquo et al., 2010). The extracts of Vernonia amygdalina and Ocimum gratissimum apart from their hypoglycemic actions could protect the heart against impairment and complete destruction due to diabetes (Asuquo et al., 2010). These plants should be used in combination as they exert a better improvement to combat the adverse effect of diabetes on the testes of male rats (Asuquo et al., 2010). The World Health Organisation estimates that 75-80% of the world's population use plant medicines either in part or entirety for health care. For many, plant medicines are a necessity, as costly pharmaceutical drugs are unaffordable; and for others, the desire to seek natural alternatives with few side effects is preferable to using conventional drugs. Hence the aim of this study was to investigate the efficacy of Vernonia amygdalina and Ocimum gratissimum on alloxan-induced diabetic rats.

MATERIALS AND METHODS

Collection of Leaves

Fresh leaves of Vernonia amygdalina and Ocimum gratissimum were collected at Ijagun, Ijebu-Ode, Nigeria and were authenticated at the Department of Botany, University of Ibadan, Nigeria.

Preparation of Plant Extracts

Fresh leaves of both plants (Vernonia amygdalina and Ocimum gratissimum) were air dried for three weeks and milled to powder with an electric blender and kept in air tight containers.

Aqueous Plant Extract

Vernonia amygdalina and Ocimum gratissimum leaf powders were soaked in distilled water for 48 hours in a desiccator, after which both were sieved using a piece of muslin cloth and Whatman filter paper. The resultant filtrate was then concentrated to dryness. Desired weights of these extract were dissolved in distilled water to form stock solutions.

Ethanolic Plant Extract

Leaf powders of Vernonia amygdalina and Ocimum gratissimum were soaked separately in 75% ethanol and left in separate desiccators for 48 hours. The extracts were then filtered into different containers using muslin cloth and Whatmann filter paper. The filtrates were then concentrated to dryness.

Experimental Animals

Sixty (60) male albino rats were purchased at animal facility in Faculty of Veterinary Medicine, University of Ibadan. They were maintained in the Biological Science's laboratory of Tai Solarin University of Education. The animals were fed with grower's mash and water ad libitum. They were then acclimatized for two weeks.

Experimental groups

Groups Treated With Ethanolic Extract

Thirty animals were divided into six groups of five animals per group. Group A (control), Group B (induced with 160mg/kg of alloxan only), Group C (induced with 160mg/kg of alloxan, and treated with 0.5mg/kg of gilbenclamide), Group D (induced with 160mg/kg of alloxan, and treated with ethanolic extract of Ocimum gratissimum at 200mg/kg), Group E (induced with 160mg/kg of alloxan, and treated with ethanolic extract of Vernonia amygdalina at 200mg/kg), Group F (induced with 160mg/kg of alloxan, and treated with combined ethanolic extract of Vernonia amygdalina and Ocimum gratissimum at 200mg/kg in ratio 1.1).

Groups Treated With Water Extract

Similar to groups treated with ethanolic extract, 30 animals were divided into six groups of five animals per group. Group A (control), Group B (received 160mg/kg of alloxan only), Group C (received 160mg/kg of alloxan and 0.1mg/kg of glibenclamide), Group D (received 160mg/kg of alloxan and 200mg/kg of Ocimum gratissimum water extract), Group E (received 160mg/kg of alloxan, and 200mg/kg of Vernonia amygdalina water extract) Group F (received 160mg/kg of alloxan and 200mg/kg of a combined water extracts of Vernonia amygdalina and Ocimmum gratissimum in ratio 1.1.).

Determination of Weight and Blood Glucose Level of Rats

A manual weighing balance was used to determine the body weight of the rats. Fasting blood glucose was determined with accucheck glucometer before the animals were fed. A drop of blood was released from the tail of rats on accucheck strip, which was inserted into the glucometer, for the observation of their blood glucose.

Statistical Analysis

Data was analyzed using one-way ANOVA in Statistical Packaging for Social Sciences (SPSS) version 16.0. The results were presented in Mean \pm Standard error and it was considered significant at P< 0.05.

RESULTS

Table 1 shows the mean fasting blood glucose of experimental rats treated with the ethanolic extracts. Prior to alloxan induction, the fasting blood glucose (FBG) levels ranged from 67 mg/dl (in combined extract) to 110mg/dl (control group). On day 2 post induction, fasting blood glucose of all the animals across the groups increased significantly (P<0.05) compared to pre induction across all the six groups. The experimental rats in groups treated with alloxan only and gibenclamide had the highest fasting blood glucose value of 600 (±0.00) mg/dl on day 2 of post induction. On day 3 post induction the fasting blood glucose of experimental rats of the control group (Group A), Ocimum gratissimum group and V. amygdalina group reduced compared to their corresponding values on day 2 post induction. Meanwhile the fasting blood glucose value of the combined extract (Group F) increased significantly (P<0.05) compared to the value obtained on day 2 post induction. The

fasting blood glucose of alloxan only and glibenclamide group remained unchanged (600 $\pm 0.00 \text{ mg/dl}$) when day 3 and day 2 post induction values were compared. On day 4 post induction, the highest glucose level was observed in alloxan only and glibenclamide group (600 $\pm 0.00 \text{ mg/dl}$); a decrease in fasting blood glucose of V. amygdalina (262 $\pm 4.41 \text{ mg/dl}$) and combined extract (556 $\pm 35.05 \text{ mg/dl}$); and an increase in fasting blood glucose of O. gratissimum group compared to day 3 post induction was observed. The control group had

the lowest fasting blood glucose of 103 (\pm 3.84) mg/dl on day 4 post induction. Marked reduction was observed in glibenclamide group, O. gratissimum and V. amygdalina and no survivor remained in alloxan only group and combined extract before day 8 post induction. However on day 8 post induction, O. gratissimum had the lowest fasting blood glucose of 81 (\pm 10.02) mg/dl when compared to other test groups while the highest fasting blood glucose of 600 (\pm 0.00) mg/dl was observed in alloxan only group.

TABLE 1: Mean blood glucose (mg/dl) of the experimental rats treated with ethanolic extract

Groups	Pre ind	Day 2pt	Day Spi	Day 4pi	Day opi	Day opi
Group A	110 ±3.51 ^c	109.67 ± 2.33^{a}	102 ± 2.85^{a}	103 ± 3.84^{a}	100 ±6.07 ^a	$98\pm\!6.35^{a}$
Group B	69.67 ± 2.96^{a}	$600 \pm 0.00^{\circ}$	$600 \pm 0.00^{\circ}$	600 ± 0.00^d	$600\pm\!0.00^d$	$600\pm\!0.00^d$
Group C	88.33 ± 7.69^{b}	$600 \pm 0.00^{\circ}$	$600 \pm 0.00^{\circ}$	$600\pm\!0.00^d$	383 ±13.78°	98.33 ± 7.27^{a}
Group D	97.67 ± 6.07^{bc}	373 ± 2.89^{b}	$286\pm\!\!34.91^b$	381 ±4.81 ^c	$210\pm\!\!56.16^{b}$	81 ± 10.02^a
Group E	87 ± 1.53^{b}	590 ± 10.00^{c}	304 ± 3.79^{b}	$262 \pm 4.41^{\text{b}}$	$264 \pm 10.21^{\text{b}}$	$295 \pm \! 30.51^{\text{b}}$
Group F	67 ±3.51 ^a	477 ± 1.17^{bc}	568 ±32.33°	556 ± 35.05^{d}	449 ± 26.77^{d}	$259 \pm 20.60^{\circ}$

Values are expressed as Mean Standard Error of the three replicates. Values in the same column followed by the same superscript are not significantly different.

Table 2 shows the mean fasting blood glucose of experimental rats treated with water extract. Prior to alloxan induction, the fasting blood glucose levels ranged from 64 (± 1.53) mg/dl (V. amygdalina) to 110 (±3.51) mg/dl (Alloxan only). On day 2 post induction the fasting blood glucose increased significantly (P<0.05) across the groups compared to pre-induction. Fasting blood sugar however reduced on day 3 post induction for control (101 ± 2.85 mg/dl), O. gratissimum (286 ± 34.91 mg/dl) and V. amygdalina (300 ±3.79 mg/dl) compared to their corresponding values obtained on day 2 post induction (alloxan only-109.67 ±4.36 mg/dl, O. gratissimum- 450.33 ±49.66 mg/dl, and V. amygdalina - 449.33 ±16.18 mg/dl). Meanwhile alloxan only (596 $\pm 0.00 \text{ mg/dl}$), glibenclamide (500 $\pm 0.00 \text{ mg/dl}$) and the combine extract all increased fasting blood glucose significantly (P<0.05) compared to their corresponding day 2 post induction values. On day 4 post induction only the combined extract group caused reduction in the fasting blood glucose with a value of 548.33 (\pm 7.54) mg/dl compared to 550 (\pm 32.33) mg/dl which was obtained on day 3 post induction. The alloxan only, glibenclamide, O. gratissimum and V. amygdalina groups all showed increased fasting blood glucose on day 4 post induction compared to what was obtained on day 3 post induction. On day 6 post induction there was significant reduction (P<0.05) in fasting blood glucose across all the groups compared to day 4 post induction. The control group recorded the lowest fasting blood glucose value of 100 (±6.07) mg/dl while the alloxan only group had the highest fasting blood glucose value of 590 (± 0.00) mg/dl on day 6 post induction. On day 8 post induction the control group, glibenclamide group, O. gratissimum group, all recorded reduced fasting blood glucose values of 98 (±6.35) mg/dl, 98.33 (±7.27) mg/dl, 105.67 (±10.02) mg/dl, 305.67 (±68) mg/dl respectively compared to values obtained on day 6 post induction.

Groups	Pre ind	Day 2pi	Day 3pi	Day 4pi	Day 6pi	Day 8pi
Group A	$110\pm\!\!3.51^d$	109.67 ±4.36 ^a	101 ± 2.85^{a}	$102.6\pm\!\!3.84^a$	100 ± 6.07^{a}	98 ±6.35 ^a
Group B	69.67 ± 2.9^{a}	593 ± 7.00^{b}	$596 \pm 0.00^{\circ}$	600 ± 0.00^{c}	590 ± 0.00^d	$600\pm\!\!0.00^d$
Group C	$88.33 \pm 7.69^{\circ}$	131.67 ± 17.82^{a}	$500 \pm 0.00^{\circ}$	$600 \pm 0.00^{\circ}$	$380 \pm 13.78^{\rm c}$	98.33 ± 7.27^{a}
Group D	82 ± 3.98^{b}	450.33 ± 49.66^{b}	$286\pm\!\!34.91^b$	311 ± 78.70^{b}	$200 \pm \! 56.16^{b}$	105.67 ± 10.02^{a}
Group E	64 ± 1.53^{a}	$449.33 \pm\! 16.18^{b}$	300 ± 3.79^{b}	600 ± 0.00^{c}	$260\pm\!\!10.21^{b}$	$412.67 \pm 39.43^{\circ}$
Group F	85.33 ± 3.98^{b}	362.00 ± 32.2^{b}	$550 \pm 32.33^{\circ}$	$548.33 \pm 7.54^{\circ}$	440 ± 26.77^{d}	305.67 ± 68^{b}

TABLE 2: Mean blood glucose (mg/dl) of the experimental rats treated with water extract

Values are expressed as Mean Standard Error of the three replicates. Values in the same column followed by the same superscript are not significantly different.

Table 3 shows the mean body weight of experimented animals treated with ethanolic extract. Prior induction with alloxan, the body weight ranged from 118 (± 0.88) g (control) to 211 (± 1.76) g (in alloxan only). On day 2 post induction, there was a reduction in the weight of animals in all the groups, except control which showed an increase in the weight of animals. This trend remains the same on day 3 post-induction, except in O. grattissmum where the weight remains unchanged, when compared with day 2 post induction. On day 4 post induction, a significant decrease in the body weight can be observed in all groups, except control group. On day 6 post induction, the result show that an increase in the body weight of animals can be observed in control and O. gratissimum; a decrease in the weight of the other test groups. On day 8 post induction, a significant increase in body weight of animals with O. gratissimum and control can be observed, and a decrease in the body weight of other test groups.

Table 3: M GROUPS	ean body weigh PREIND	nt (g) of the anin DAY 2 PI	nals in the vario DAY 3 PI	us experimental DAY 4 PI	l groups (ethano DAY 6 PI	olic extract) DAY 8 PI
Group A	118±0.88 ^a	121.57±0.83 ^{ab}	123.93±1.13 ^{ab}	129.00±2.73 ^b	135.97±4.83°	141±5.49 ^b
Group B	211 ± 1.76^d	207.33±1.47 ^e	$201.33{\pm}2.60^{d}$	200.13 ± 2.91^{d}	1.93±2.52 ^e	188.07 ± 1.82^{d}
Group C	141 ± 1.20^{b}	135.37±0.88 ^c	$130.93{\pm}1.35^{b}$	126±2.13 ^{ab}	110.8 ± 4.26^{a}	103.03±0.65 ^a
Group D	121 ± 2.52^{a}	177.97±2.55 ^a	177.5 ± 0.76^{a}	120±1.66 ^a	126.27 ± 1.79^{b}	135.13±1.31 ^b
Group E	$134.37 {\pm} 4.88^{b}$	127.4 ± 3.51^{b}	122.53±2.05 ^a	119.33±2.50 ^a	111.03±0.58 ^a	106.3±1.14 ^a
Group F	$194 + 1.73^{\circ}$	$187.47 + 3.65^{d}$	182.93+4.34 ^c	$179+2.74^{\circ}$	$173.4 + 1.21^{d}$	$173.17 \pm 1.96^{\circ}$

Group F 194 ± 1.73^{c} 187.47 ± 3.65^{d} 182.93 ± 4.34^{c} 179 ± 2.74^{c} 173.4 ± 1.21^{d} 173.17 ± 1.96^{c} Values are expressed as Mean Standard Error of the three replicates. Values in the same column followed by the same superscript are not significantly different.

In Table 4, prior to alloxan induction, the body weights ranged from 118 (± 0.88) g (control group) to 211 (± 1.76) g (alloxan only group). There was significant reduction (P<0.05) in the body weight of the experimental animals across the groups compared to pre induction. On day 3 post induction the control, O. gratissimum, and combined extract groups all had higher body weight values of 121.93 (± 1.13) g, 178.5 (± 0.76) g, and 180.93 (± 4.34) g compared to their corresponding values obtained on day 2 post induction. Meanwhile on day 4 post induction all the groups except V. amygdalina group had reduced mean body weight values of 120 (± 2.73) g on day 4 post induction. On day 6 post induction the reduced body weight values of all the groups except the control and combined extract group also had reduced body weight values compared to day 4 post induction. The lowest mean body weight values of all the groups except the control and combined extract group also had reduced body weight values compared to day 4 post induction. The lowest mean body weight values of all the group) was obtained on day 6 post induction. On day 8 post induction the reduced body weight values compared to day 4 post induction. The lowest mean body weight value of 109.8 (± 4.26) g (glibenclamide group) was obtained on day 6 post induction. On day 8 post induction there was significant reduction (P<0.05) in the mean body weight values of the animals in alloxan only, glibenclamide, and combined extract groups.

Table 4: M	ean body	weight (g) of the	animals in the	various experin	mental groups ((water extract)
CDOUDC	DDEIND	DAVADI		DAV 4 DI	DAVCDI	DAVODI

GROUPS	PREIND	DAT 2 PI	DAISPI	DAT 4 PI	DAIOPI	DAIOPI
Group A	118±0.88 ^a	121.57±0.83 ^a	121.93±1.13 ^{ab}	120±2.73 ^a	135.97±4.83°	141±5.49 ^c
Group B	211 ± 1.76^{b}	207.33±1.47 ^e	$200.33{\pm}2.60^d$	200.13 ± 2.91^{d}	193±2.52 ^e	$188.07{\pm}1.82^{d}$
Group C	141±1.20 ^c	$135.37{\pm}0.88^{b}$	132.93±1.35 ^b	126±2.31 ^a	109.8±4.26 ^a	103.03±0.65 ^a
Group D	152 ± 2.04^d	150±2.16 ^c	178.5±0.76 ^a	140 ± 0.89^{b}	$124.27{\pm}1.79^{b}$	131±1.82 ^b
Group E	163±1.04 ^e	159±0.16 ^{cd}	120.53±2.05 ^a	140±2.89 ^b	110.03±0.58 ^a	132.5 ± 2.82^{b}
Group F	170±1.67 ^e	162 ± 0.67^{d}	180.93±4.34 ^c	150.4±0.67 ^c	170.4 ± 1.21^{d}	138.9 ± 1.17^{bc}

Values are expressed as Mean Standard Error of the three replicates. Values in the same column followed by the same superscript are not significantly different.

Table 5 shows the weight gain or loss of experimental rats prior alloxan induction and day 8 post induction. At day 8 post induction, there was increase in body weight of experimental animals (weight gain) in control group and O. gratissimum extract respectively. A drastic reduction in body weight of animals in groups treated with V. amygdalina, combined extract, gilbenclamide and alloxan only can be observed.

Table 5: Weight gain/loss of alloxan-induced diabetic rats on day 8 post induction (ethanolic extract)GROUPSW1(g)W2 (g)WG (g)WL (g)

Group A	118	141	23	-
Group B	211	188.07	-	22.93
Group C	141	103.33	-	37.97
Group D	121	135.13	14.13	-
Group E	134.37	106.30	-	28.07
Group F	194	173.17	-	20.83

In Table 6 there was weight loss in the alloxan only, glibenclamide, V. amygdalina and combined extract groups. The combined extract group had the lowest weight loss of 20.83g compared to all other groups.

Table 6: Weight gain/loss of alloxan-induced diabetic rats on day 8 post induction (Water extract) GROUPS W1(g) W2 (g) WG (g) WL (g)

Group A	118	141	23	-
Group B	211	188.07	-	22.93
Group C	141	103.03	-	37.97
Group D	152	121	-	31
Group E	163	132.5	-	30.5
Group F	170	138.9	-	31.9

DISCUSSION

In this study, Vernonia amygdalina reduced the glucose level in diabetic rats, but its action was slower than that of glibenclamide and O. gratissimum. This could be as a result of the route of administration of the extract. The cause of the slow reaction of the extract is yet unknown. However, the result still agrees with Modu et al. (2013) who investigated on the glycemic effect of amygdalina in combination with V. а hypoglycemic drug (metformin). They reported that the V. amygdalina extract alone reduced the glycemic significance in the alloxan treated rats and lends credence that it has peripheral action similar to that of insulin or glucose metabolism which can be attributed to the bioactive molecules present in the indigenous vegetable. Akah et al. (2004) evaluated the effect of V. amygdalina aqueous leaf extract on serum glucose and triglyceride level of diabetic rats, and they reported that the extract caused significant and progressive time dependent reduction of blood glucose and serum triglyceride in normoglycemic and alloxan induced diabetic rats. Likewise, Mfon et al. (2011) evaluated the effect of combined extracts of V. amygdalina and Gongronema latifolium on the pancreas of streptozotocin induced diabetic rats and they reported that the combined extract has hypoglycaemic effect. Additionally, Chike et al. (2006) determined the aqueous leaf extract of V. amygdalina on blood glucose on alloxan induced diabetic rats, and also agreed that it has hypoglycemic potential.

However, the result of this study showed that extract of O. gratissimum was more effective, as it showed a higher potential of reducing the fasting blood glucose of experimental animals. It proved to be more potent, and at a faster rate. This suggests that O. gratissimum has some active components which makes it more effective than V. amygdalia which makes it more effective than the other. This agrees with the findings of Asuquo et al. (2010) who investigated the ethanolic extracts of V. amygdalina and O. gratissimum to enhance testicular improvement in diabetic Wistar rats, and claimed that O. gratissimum had a more positive and potent effect on the testes of diabetic rats compared to V. amygdalina. He attributed it to the high level of alkaloids found in O. gratissimum. Similarily, Opara et al. (2012) evaluated the hematological and biochemical responses of adult rabbits when given aqueous extract of Ocimum gratissimum leaves, and they reported that aqueous extract of O. gratissimum leaves can be used up to 40% level in drinking water for adult rabbits to increase immunity and serum protein levels, beside other hematological and biochemical benefits, without any deleterious effects on the animals. These results showed that O. gratissimum can be useful in elevating the blood glucose level of diabetic mammals. Nworgu et al. (2013) in their findings concluded that O. gratissimum is a potent hypoglycemic supplement for growing pullets. The result also showed that glibenclamide reduced the elevated blood glucose of the diabetic rats better. This corroborates the

report of Erejuwa et al. (2011), who reported that glibenclamide has the ability to reduce the elevated blood glucose of diabetic rats, but that it could not improve the body weight in those rats. In this study the ethanolic extracts of O. gratissimum, V. amygdalina and the combined extracts were able to reduce fasting blood glucose and increase the body weights of the experimental rats better than water extract treatment. Weight loss is one of the symptoms of diabetes mellitus occurring especially when glycemic control is poor. Studies have equally reported significant weight reduction in untreated diabetic rats (Mfon et al., 2011). At the end of the research, there was a significant increase in the body weight of the rat treated with O. gratissimum and control. This disagrees with the report of Arhoghro et al. (2012), who investigated the curative potential of aqueous extract of O. gratissimum on cisplatin induced hepatotoxicity in albino rats.

Conclusion

The ethanolic extract of both O. gratissimum and V. amygdalina can be used separately to reduce the fasting blood glucose of diabetic rats. O. gratissimum has more efficacy than glibenclamide, and can also be helpful in restoring reduced body weight of these animals.

REFERENCES

Akah P, Njoku O, Nwanguma A, Akunyili D (2004). Effects of Aqueous of Vernonia amygdalina on Blood Glucose and Triglyceride Level of Alloxan Induced Diabetic Rats(Rattus rattus). Animal research 1 (2): 90- 94.

Arhoghro E, Ikeh C, Uwakwe A, Ekpo K, Anosike E (2012). Curative Potential of aqueous extract of scent leaf (Ocimum gratissimum) on cisplatin induced hepatotoxicity in albino wistar rats. Pharmaceutical and Scientific innovation 1(1).

Asuquo O, Igiiri AO, Akpan JE, Akapaso MI (2010). Cardio protective Potential of Vernonia amygdalina and Ocimmum gratissimum against streptozoticin (STZ) induced diabetes Wistar rats. Tropical Medicine, 7(1).

Chike CP, Georgewill OA, Nnodi CU (2006). Effect of Aqueous Leaf Extract of Vernonia amygdalina (bitter leaf) on blood glucose concentration of Alloxan induced Diabetic Albino Wistar Rats. Applied Zoology and Environmental Biology, 8: 44-47.

Chukwuma M (2012). Bitter leaf, Scent leaf extract protects Diabetics from Heart, Testicular Damage. Guardian. 19 July, 2012.

Efiri EC (2012). The effect of Ocimum gratissimum on the histology of the lungs of albino rats.

The benefits of herbs O. gratissimum. Scent leaf, Aug 10.

Erejuwa OO, Suliaman SA, Wahab MS, Gurtu S (2011). Effect of Glibenclamide alone

versus Glibenclamide and Honey on oxidative stress in Pancreas of Streptozotocin induced Diabetic rats. Nature products, 4(2): 1-10.

Mfon I, Item J, Amabe A, Victor A, Anozeng O, Patrick E (2011). Effects of combined leaf extract of Vernonia amygdalina (bitter leaf) and Gogronema latifolium (Utazi) on the pancreatic B- cells of streptozotocin induced diabetic rats. Medicine and medical research, 1(1): 24-34

Modu S, Adeboye AE, Maisaratu A, Mubi BM (2013). Studies on the Administration of Vernonia amygdalina Del (bitter leaf) and Glucophage on Blood Glucose level of alloxan induced Diabetic rats. Medicinal plants and administrative medicine, 1(1): 13-19.

Nworgu FC, Yekini BO, Oduolai OA (2013). Effects of Basil leaf (Ocimum gratissimum) supplement on some blood parameters of growing pullets.

Opara MNN, Iwuji TC, Igwe IN, Etuk IF, Maxwell JA (2012). Haematological and Biochemical responses of Adult Rabbit to Aqueous Extract of Ocimum gratissimum leaves. Physiology and Pharmacology, 2(9): 301- 306.

Sarah W, Gojka R, Anders G, Richards S, Hilary K (2000). Global prevalence of diabetes: Estimates for the year 2000 and projections for 2030. Diabetes Care, 27(5): 1047-1053.