B-5462
[1-6]

RESEARCH ARTICLE Indian Journal of Animal Research

Effect of Ethanolic Extract of *Scurrula parasitica* L. on *in vivo* Antioxidant Status of Diabetic Rats

R. Zapaw Azyu[1], M. Ayub Ali[1], Jagan Mohanarao Gali[1], Parthasarathi Behera[1], Hemen Das[1], Prava Mayengbam[1], L. Inaotombi Devi[2]

10.18805/IJAR.B-5462

ABSTRACT

Background: *Scurrula parasitica* L. is a herbaceous growing shrub of *Loranthaceae* family traditionally used for diuretic, tranquilizing and hypotensive activity. It shows anti-diabetic, cytotoxic, anticancer, anti-hepatoxic and immunomodulatory activity as it has secondary metabolites like alkaloids, terpenoids, phenols, flavonoids, tannins, saponins, *etc.* The secondary metabolites have potential antioxidant activity as they have the ability to scavenge free radicals and also improve the *in vivo* antioxidant status. However, no study has been conducted to evaluate their effect on *in vivo* antioxidant status.

Methods: The *in vitro* antioxidant activity was estimated by three *in vitro* assay methods *viz.* DPPH free radical scavenging assay, Ferric reducing antioxidant potential (FRAP) assay and Total phenolic content (TPC) assay while the *in vivo* antioxidant status was evaluated by estimating the levels of SOD, Catalase, GPx and MDA.

Result: The ethanol extract of the leaves shows maximum *in vitro* antioxidant activity in case of DPPH free radical scavenging assay and 50% ethanol extract shows highest antioxidant activity for FRAP assay and Total phenolic content assays. The ethanol extract of the plants improves the *in vivo* antioxidant status *viz.* Superoxide dismutase (SOD), Catalase, Glutathione peroxidise (GPx) and Malondehyde (MDA) among the STZ induced diabetic rates.

Key words: Catalase, Gpx, MDA, *Scurrula parasitica* L., SOD.

INTRODUCTION

Medicinal plants are found to have many therapeutic properties against certain diseases or serve as the origin of useful drugs (Ebadi, 2006). They are used for curing many ailments as they contain phytochemicals of various biological activities (Balasubramanian *et al.,* 2014). The medicinal value of the medicinal plants is because of the secondary metabolites which have definite physiological action on the human body (Akinmoladun *et al.* 2007). The phenolic compounds such as flavonoids, phenolic acids, tannins, *etc.* have potential antioxidant activity as they have the ability to scavenge free radicals. Further, these compounds have metal chelating, antimutagenic, anticarcinogenic and antimicrobial activities (Proestos *et al.,* 2005).

Antioxidants can scavenge free radicals and can raise the level of endogenous antioxidant defence. There is dynamic balance between the amount of free radicals generated in the body and antioxidants against their deleterious effects (Finkel and Holbrook, 2000; Ali *et al.,* 2013). However, the amounts of these protective antioxidant principles present under the normal physiological conditions are sufficient only to cope with the physiological rate of free radicals, either from environment or produced within the body. In order to maintain the level of antioxidant in the body for healthy living, external supplementation is necessary (Ali *et al.,* 2013). Recent studies have investigated the potential of plant products as antioxidants against various diseases induced by free radicals. The *in vivo* antioxidant defence system consists mainly of superoxide dismutase (SOD), catalase (CAT), glutathione peroxidase (GPx) and glutathione reductase (GSSGR) (Ames *et al.,* 1993; Sardesai, 1995). These antioxidant enzymes are an important protective mechanism against Reactive Oxygen Species (ROS). Oxidative stress (OS) is essentially an imbalance between the production of free radicals, such as superoxide, hydroxyl (OH) and peroxyl (ROO) radicals and the ability of the body to counteract or detoxify their harmful effects through neutralization by antioxidants. Oxidative stress leads to many chronic and degenerative diseases such as cancer, atherosclerosis, Parkinson's disease, Alzheimer's disease, diabetes, neurodegenerative disorders and aging (Yu, 1994; Choudhary and Tandon, 2009). As the antioxidant enzymes neutralize the reactive oxygen species in the body, higher level of *in vivo* antioxidant enzymes would lead to higher

[1]College of Veterinary Sciences and Animal Husbandry, Central Agricultural University, Selesih, Aizawl-796 001, Mizoram, India.
[2]Department of Medical Laboratory Science, Regional Institute of Paramedical and Nursing Sciences, Aizawl-796 001, Mizoram, India.

Corresponding Author: M. Ayub Ali, College of Veterinary Sciences and Animal Husbandry, Central Agricultural University, Selesih, Aizawl-796 001, Mizoram, India.
Email: ayubali1969@gmail.com

How to cite this article: Azyu, R.Z., Ali, M.A., Gali, J.M., Behera, P., Das, H., Mayengbam, P. and Devi, L.I. (2025). Effect of Ethanolic Extract of *Scurrula parasitica* L. on *in vivo* Antioxidant Status of Diabetic Rats. Indian Journal of Animal Research. 1-6. doi: 10.18805/IJAR.B-5462.

Submitted: 01-08-2024 **Accepted:** 09-02-2025 **Online:** 14-05-2025

protection of the body organs from the degenerative diseases.

Scurrula parasitica L. is a herbaceous growing shrub of *Loranthaceae* family (Bambaradeniya *et al.,* 2002). It is a parasitic plant grown on *Dendrophthoefalcata* and on a wide range of hosts, including species of *Apocynaceae, Euphorbiaceae, Fabaceae, Fagaceae, Lythraceae, Moraceae, Punicaceae, Rosaceae, Rutaceae, Sapindaceae, Theaceae* and *Ulmaceae*. Traditionally it is used as diuretic, tranquilizing and hypotensive drug (Haque *et al.,* 2016). *Scurrula parasitica* plant shows anti-diabetic, cytotoxic, anticancer, anti-hepatoxic and immunomodulatory activity (Mahajan *et al.,* 2013). Phenolic compound of *Scurrula parasitica* L. has important phytochemicals which possess anti-inflammatory, antiallergic, antithrombotic, antimicrobial and anticancerous and analgesic activities. (Purneetha and Amruthesh, 2016). Though many studies have been conducted on the medicinal properties of *Scurrula parasitica* L., the effects of this plant on the *in vivo* antioxidant enzymes have not been reported so far. In view of this, the present research work is planned to investigate the *in vitro* antioxidant capacity of the *Scurrula parasitica* L. extracts in different solvent and also the effect of ethanol extract on *in vivo* antioxidant status.

MATERIALS AND METHODS

The experiment was carried out in the department of Veterinary Physiology and Biochemistry, College of Veterinary Sciences and Animal Husbandry, Central Agricultural University, Selesih, Aizawl, Mizoram, India during March to November, 2022. The use of experimental animals and the experimental protocol has been duly approved by the institutional Animal Ethics Committee of the college vide No. CVSC/CAU/IAEC/21-22/P-8 dated, the 16th November, 2022. Internationally acceptable methods/techniques/ protocols were followed during the present investigation and all the chemical/ drugs used were of high technical grades.

Plant collection and authentication

The plants of *Scurrula parasitica* L. were collected from Mualvum, Kawnpui, Kolasib district of Mizoram, India.

Chemicals and reagents

Streptozotocin, Metformin, Chloroform and Tween 80 were purchased from HiMedia Laboratories Pvt. Ltd., Ethanol, Petroleum ether, Methanol, Ferric Chloride, Sodium Potassium Tartarate, Folin Ciocalteu reagent were purchased from Merck Limited. 1,1-Diphenyl-2-picrylhydrazyl (DPPH), 2,4,6-tri(2-pyridyl)-striazine (TPTZ), 6-hydroxy-2,5,7,8-tetramethyl chromane-2- carboxylic acid (Trolox) and Gallic acid from Sigma Chemicals Co. (St. Louis, USA). The kits for estimation of *in vivo* antioxidant enzymes *viz.* SOD, Catalase, GPx, MDA were from Wuhan Fine Biotech Co. Ltd. All the glassware used were of borosilicate quality.

Wistar rat

Adult Wistar rats were procured from M/s. Ruata Enterprise, Aizawl. Animals were housed in polypropylene cages in small groups of 6 rats per cage. Animals had free access to standard balanced ration and clean drinking water *ad-libitum* and were maintained in standard laboratory conditions (12:12 hour light/dark cycle at ambient temperature ranging between 20-25°C).

Processing of material

The *Scurrula parasitica* L. leaves were washed in running tap water and finally rinsed with distilled water. The leaves were air dried in shade till completely dried and then ground to powder by a mechanical grinder and then soaked in solvents (1:5 w/v) with intermittent stirring for 5 days at room temperature. The filtrate was concentrated under reduced pressure as per the method described by Zakaria *et al*. (2004) till the extract appears sufficiently dry and stored at -20°C.

In-vitro antioxidant assay

The *in vitro* antioxidant content of the extracts was estimated by three *in vitro* assay methods *viz.* DPPH free radical scavenging assay as described by Leong and Shui (2001), Ferric reducing antioxidant potential (FRAP) assay according to the procedure of Benzie and Strain (1999) and Total phenolic content (TPC) assay by the Folin-Ciocalteau method of Singleton and Rossi (1965).

Preparation of oral suspension

The ethanol extract of the *Scurrula parasitica* L. leaves was dissolved using 2% Tween 80. As per the requirement, 10% (100mg.ml^{-1}) and 20% (200mg.ml^{-1}) solutions were prepared for oral administration.

Dose rate for evaluation of *in vivo* antioxidant activity

The dose rate of (100 mg/bw kg^{-1} and 200 mg/bw kg^{-1}) for evaluation of *in vivo* antioxidant status were chosen as reported by Laldingngheta *et al*. (2019).

Diabetes induction

Thirty healthy male Wistar rats were selected and divided into 5 groups of 6 animals each. The rats were kept off the feed for 24 hours and then the rats except from Group-I was administered streptozotocin at a dose of 40mg.kg-1 intraperitoneally as per the method of Kalaivanan and Pugalendi (2011). The rats which have blood glucose level of 250 mg/dl or more after 72 hours of administration of streptozotocin were considered as diabetic rats. The Group-I rats served as normal control while Group II rats served as untreated diabetic control and received vehicle only. Rats in Group III received the standard drug, metformin at dose rate of 5 mg/kg body weight. Group IV and Group V rats received plant extracts at the dose rate of 100 mg/kg b.w. and 200 mg/kg b.w.

Assessment of *in vivo* antioxidant status

The blood samples of the rats in different groups were collected on 24th day of treatment and the *in vivo* antioxidant status of the rats were estimated. The levels of the Superoxide dismutase (SOD), Catalase, Glutathione

peroxidise and Malondehyde (MDA) of the rats were estimated with kits manufactured by M/s. Wuhan Fine Biotech, Co. Ltd. The SOD, Catalase and GPX were estimated by Sandwich enzyme immunoassay. The microtiter plate was pre-coated with antibody specific to SOD, CAT and GPX respectively. Briefly, 100 µL each of working solution or samples were added into appropriate wells, covered with plate cover and incubated for 80 min at 37°C. Pour out the liquids and washed three times with 200 µL wash solutions. Added 100 µL Biotinylated Antibody working solution in each well, covered with plate cover and incubated for 50 min at 37°C. Aspirated and washed 3 times. Added 100 µL Streptavidin-HRP working solution in the wells, covered with plate sealer and incubated for 50 min at 37°C followed by aspiration and 5 times washing. Added 90 µL of TMB substrate solution to the wells, covered with plate cover and incubate for 20 min at 37°C. Added 50 µL stop reagent and took reading at 450 nm in ELISA plate reader. The MDA was estimated by competitive inhibition enzyme immunoassay technique. Briefly, added 50 µL of standard reagent or samples into the wells and 50 µL Biotinylated-conjugate in each well, mixed well, covered with plate cover and incubated for 60 min at 37°C. Poured out the liquid, aspirated and washed with 200 µL wash solution. Added 100 µL Streptavidin- HRP working solution in the wells, covered with plate sealer and incubated for 60 min at 37°C followed by aspiration and 5 times washing. Added 90 µL of TMB substrate solution to the wells, covered with plate cover and incubate for 20 min at 37°C. Added 50 µL stop reagent and took reading at 450 nm in ELISA plate reader.

RESULTS AND DISCUSSION

The *in vitro* antioxidant status of the leaves of *Scurrula parasitica* L. extracted in different solvents and the effect of the ethanol extract on the *in vivo* antioxidant status of the STZ induced diabetic rates were evaluated. The *in vitro* antioxidant activity of the extracts was estimated by assessing the DPPH free radical scavenging activity, Ferric Reducing Antioxidant Potential (FRAP) assay and Total Phenolic content assay. The antioxidant activity of the different extracts is presented in Table 1 and Fig 1.

The ethanol extract showed comparatively higher DPPH free radical assay of 4.299±0.02 mg TE while the FRAP assay and total phenolic content assay shows highest activity in 50% ethanol extract. The observed activity in FRAP assay and total phenolic content in 50% ethanol extract were 0.482±0.01 mg TE and 24.489±1.41mg GAE respectively. The high antioxidant content of the *Scurrula parasitica* L. leaves were also reported in previous studies (Ali *et al.,* 2013; Atun *et al.,* 2017; Muhammad *et al.,* 2019, Laldingngheta *et al.* 2020). Atun *et al*. (2017) observed that the leaves of the plant *S. parasitica* contain quercitrin (quercetin-3-O-rhamnoside) which showed high antioxidant activity. Laldingngheta *et al*. (2020) also reported high antioxidant activity *i.e.* high total phenolic content and DPPH radical scavenging activity. The total phenoilc content of

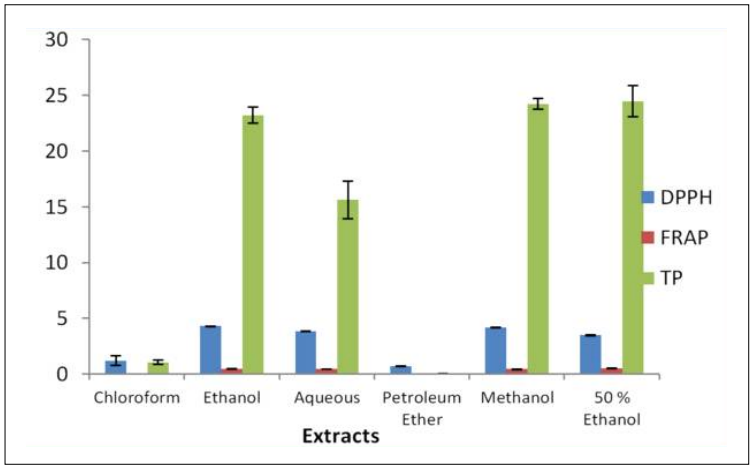

Fig 1: Antioxidant activity of the *Scurrula parasitica* L. leaves extract in different solvents.

Table 1: Antioxidant activity of the *Scurrula parasitica* L. leave extract in different organic solvents and water.

Extracts	DPPH (mgTE/gm)	FRAP (mgTE/gm)	Total phenolic content(mg GAE/gm)
Chloroform	1.197±0.43	0.00±0.00	1.053±0.19
Ethanol	4.299±0.02	0.482±0.01	23.227±0.70
Aqueous	3.850±0.03	0.465±0.00	15.623±1.70
Petroleum Ether	0.734±0.02	0.00±0.00	0.039±0.01
Methanol	4.188±0.01	0.459±0.01	24.224±0.48
50% Ethanol	3.485±0.04	0.513±0.02	24.489±1.41

the ethanolic extract estimated was around 101.0 to 379.1 (µg/gm tissue) at concentrations from 5 to 100 µg/ml plant extract. As for DPPH, the value of IC50 was found to be 53.28 µg/ml (Laldingngheta *et al.,* 2020). The high antioxidant activity of the plant extract may be due to high content of polyphenolic compounds, flavonoids *etc.* (Okudu *et al.,* 1994; Laldingngheta *et al.,* 2020).

The antioxidant activity of the plant extract in all the three methods is more or less the highest in ethanol extract therefore ethanol extract was used for evaluation of *in vivo* antioxidant status. The *in vivo* antioxidant status *viz*.. Superoxide dismutase, Catalase, Glutathione peroxidise and Malondialdehyde of the rats is altered on induction of diabetes. Treatment of the diabetic rats with metformin or the ethanol extract improves the *in vivo* antioxidant status. The *in-vivo* antioxidant profile of Streptozotocin induced Wistar rats treated with *Scurrula parasitica* L. extract is given in Table 2.

The level of malondialdehyde increased on induction of diabetes. The observed level of MDA in normal rats was 140.00±10.00 ng/mL and the level increased to 410.00±20.81 ng/mL among the diabetic group. Treatment of the diabetic rats with metformin or the plant extract significantly reduces the level of MDA. The level observed for metformin, 100 mg and 200 mg/kg b.wt plant extract were 141.67±6.00 ng/mL, 160.00±15.27 ng/mL and 170.00±15.27 ng/mL, respectively.

The level of catalase, GPx and SOD decreases on induction of diabetes. The catalase activity among normal rats was 2166.70±0.02 U/mL and decreased to 1300.00±0.01 U/mL on induction of diabetes. Treatment with metformin or 100 mg or 200 mg/kg b.wt plant extract significantly increases the activity to 1933.30±0.02 U/mL, 1733.33±0.02 U/mL and 2117.30±73.20 U/mL respectively. The GPx activity among the normal rats was 2050.00±0.01 U/mL which decreased to 1633.30±33.30U/mL on induction of diabetes. Treatment with metformin or 100 mg or 200 mg/kg b.wt plant extract significantly increases the activity. The activity level among the metformin or 100 mg or 200 mg/kg b.wt plant extract treated groups were 2033.30±44.09 U/mL, 1766.70±0.01 U/mL and 1993.30±29.62 U/mL, respectively. The Superoxide Dismutase activity among normal rats was 2.12±0.02 U/mL and the level decreased to 1.80±0.00 U/mL in case of diabetic rats. The activity for metformin or 100 mg or 200 mg/kg b.wt plant extract treated groups were 2.57±0.17U/mL, 1.9±0.02U/mL and 2.02±0.03 U/mL respectively.

Hyperglycemia in diabetes can increase production of free radicals through Amadori rearrangement (Giugliano *et al.,* 1996). Increased oxidative stress is observed in diabetic subjects (Waggiallah and Alzohairy, 2011; West, 2000; Jakus, 2000; Ceriello, 1997) and this is linked to reduced enzymatic and non-enzymatic antioxidants (Lapena *et al.,* 2018). Peroxidation of lipids produces highly reactive aldehydes, including MDA, acrolein, 4-hydroxynonenal (HNE), 4-oxononenal (ONE) and isolevuglandins (IsoLGs) (Guo *et al.,* 2012). The increased in the level of MDA in diabetes is reported in literature (Mahreen *et al.,* 2010; Shawki *et al.,* 2021; Moussa, 2008; Bandeira *et al.,* 2012). Increased level of MDA in diabetics suggests that peroxidative injury may be involved in the development of diabetic complications. It is also an indication of decline in defense mechanisms of enzymatic and nonenzymatic antioxidants (Saddala *et al.,* 2013). The depletion of GSH impairs the activity of antioxidant enzymes as well as that of chain breaking aqueous and lipid phase antioxidants (Bhatia *et al.,* 2003). The decreased level of blood SOD in diabetes is also reported in literature (Bhatia *et al.,* 2003; Blum and Fridovich, 1985; Sundraram *et al.,* 1996; Kumawat *et al.,* 2013). The reduction in serum SOD activity could be due to excessive consumption in the autoxidation process and increased excretion from the inflammatory kidney in nephropathy and also could be linked to progressive glycation of enzymatic proteins. About 50% of SOD in erythrocytes of diabetic patients is glycated, resulting in low activity of SOD (Arai *et al.,* 1985). The decreased serum catalase in diabetes is also reported in literature (Goth *et al.* 2006; Tiedge *et al.,* 1997; Syyeda Anees, 2014).

Several plant extracts have positive effect on the serum antioxidant enzymes of diabetic rats (Sani *et al.,* 2012; Dhalwal *et al.,* 2008; Kukic *et al.,* 2008). Flavonoids in plant extracts are very effective in reducing the lipid peroxidation in hypercholesterolaemic rats (Mateos *et al.,* 2005). The beneficial effect of flavonoids may be mediated by one or more mechanisms such as by inhibiting lipid per-oxidation, platelet aggregation and enhancing of antioxidant defense (Mateos *et al.,* 2005; Lin *et al.,* 1998). Further, the other

Table 2: *In vivo* Antioxidant profile of Streptozotocin induced Wistar rats treated with *Scurrula parasitica* L. extract.

Antioxidant enzymes	Treatment groups					P value
	Control	Diabetic	Metformin Treated	100 mg/Kg body weight	200 mg/kg body weight	
MDA (ng/mL)	140±10.00^{a}	410±20.81^{b}	141.67±6.00^{a}	160±15.27^{a}	170±15.27^{a}	0.00**
Catalase (U/mL)	2166.7±0.02^{b}	1300±0.01^{a}	1933.3±0.02^{b}	1733.3±0.02^{ab}	2117.3±73.2^{b}	0.04*
SOD (U/mL)	2.12±0.02^{b}	1.8±0.00^{a}	2.57±0.17^{c}	1.9±0.02^{ab}	2.02±0.03^{ab}	0.00**
GPX (pg/mL)	2050±0.01^{b}	1633.3±33.3^{a}	2033.3±44.09^{b}	1766.7±0.01^{ab}	1993.3±29.62^{b}	0.02*

Values are mean±SEM, n=6 in each group. NS:Non-significant.*:P<0.05, **:P<0.01.The means with different superscript in small letters differs significantly (P≤0.05) between the columns.

secondary metabolites *i.e.* polyphenols such as phenolic acids, proanthocyanidins are potent antioxidants. They act as chain breaking antioxidants, meaning that they can directly interact with and neutralize lipid radicals, halting the propagation of lipid peroxidation (Zhu *et al.,* 2013), thereby preventing the formation of harmful lipid peroxidation products like malondialdehyde (MDA) (Gorelik *et al.,* 2008). Thus polyphenols might be responsible for suppressing the extent of lipid peroxidation and enhancing the antioxidant capacity in the liver. In the present investigation, the improvement in serum antioxidant status of the diabetic rats on treatment with extract of *Scurrula parasitica* L. may be due high content of secondary metabolites like alkaloids, flavonoids, terpenoides, phenols, tannins, saponins, curcumins *etc.*

CONCLUSION

The leaves of *Scurrula parasitical* L. have high *in vitro* antioxidants. Ethanol extract shows highest activity in DPPH free radical scavenging activity while highest activity in ferric reducing antioxidant potential (FRAP) assay and total phenolic content assay was observed in 50% ethanol extract. Diabetes reduces the antioxidant level of the body. The level of SOD, Catalase, GPx decreased while the level of MDA was increased on induction of diabetes. Flavonoids and other plant secondary metabolites are very effective in reducing the lipid peroxidation by inhibiting lipid per-oxidation, platelet aggregation and enhancing of antioxidant defense. Thus treatment with *Scurrula parasitica* L. extract improves the *in vivo* antioxidant status.

ACKNOWLEDGEMENT

We are thankful to the Dean, College of Veterinary Sciences and A.H., Central Agricultural University, Selesih, Aizawl for providing all the required materials and the facility required to conduct this research work.

Conflict of interest

All authors declared that there is no conflict of interest.

REFERENCES

Akinmoladun, A.C. Ibukun, E.O. Afor, E. Akinrinlola, B.L. Onibon, T.R. Akinboboye, A.O. and Farombi, E.O. (2007). Chemical constituents and antioxidant activity of Alstoniaboonei. African J. Biotechnol. 6(10): 197-1201.

Ali, M.A. Chanu, K.V. and Devi, L.I. (2013). *Scurrula parasitica* L. A medicinal plant with high antioxidant activity. Int. J. Pharm Pharm Sci. 5(1): 34-37.

Ames, B.N. Shigenaga, M.K. and Hagen, T.M. (1993). Oxidants, antioxidants and the degenerative diseases of aging. Proc. Natl. Acad. Sci. U.S.A. 90(17): 7915-7922.

Arai, K. Maguchi, S. Fujii, S. Ishibashi, H. Oikawa, K. and Taniguchi, N. (1987). Glycation and inactivation of human Cu-Zn-superoxide dismutase. Identification of the *in vitro* glycated sites. J. Biol. Chem. 262(35): 16969-16972.

Atun, R. Davies, J.I. Gale, E.A.M. Bärnighausen, T. Beran, D. Kengne, A.*P. et al.* (2017). Diabetes in sub-Saharan Africa: from clinical care to health policy. The Lancet. Diabetes and Endocrinology. 5: 622-667.

Balasubramanian, S. Ganesh, D. and Narayana, S. (2014). GC-MS analysis of phytocomponents in the methanolic extract of Azadirachtaindica (*Neem).* Int. J. Pharma. Bio. Sci. 5(4): 258-262.

Bambaradeniya, C.N. Ekanayake, S.P. Kekulandala, L.D.C.B. Fernando, R.H.S.S. Samarawickrama, V.A.P. and Priyadharshana, T.G.M. (2002). An assessment of the status of biodiversity in the Maduganga mangrove estuary. Occ. Pap. IUCN, Sri Lanka. 1: 49.

Bandeira, S.D.M. Guedes, G.D.S. Fonseca, L.J.S.D. Pires, A.S. Gelain, D.P. Moreira, J.C.F. and Goulart, M.O.F. (2012). Characterization of blood oxidative stress in type 2 diabetes mellitus patients: increase in lipid peroxidation and SOD activity. Oxid. Med. Cell. Longev. 2012: 1-13. doi:10.1155/2012/819310

Benzie, I.F. and Strain, J.J. (1999). Ferric reducing/antioxidant power assay: direct measure of total antioxidant activity of biological fluids and modified version for simultaneous measurement of total antioxidant power and ascorbic acid concentration. Meth. Enzymol. Academic Press. 299: 15-27.

Bhatia, S. Shukla, R. Madhu, S.V. Gambhir, J.K. and Prabhu, K.M. (2003). Antioxidant status, lipid peroxidation and nitric oxide end products in patients of type 2 diabetes mellitus with nephropathy. Clin. Biochem. 36(7): 557-562.

Blum, J. and Fridovich, I. (1985). Inactivation of glutathione peroxidase by superoxide radical. Arch. Biochem. Biophys. 240(2): 500-508.

Ceriello, A. (1997). Acute hyperglycaemia and oxidative stress generation. Diabet. Med. 14(S3): S45-S49.

Choudhary, R. and Tandon, R.V. (2009). Consumption of functional food and our health concerns. Pak. J. Physiol. 5(1): 76-83.

Dhalwal, K. Shinde, V.M. Namdeo, A.G. and Mahadik, K.R. (2008). Antioxidant profile and HPTLC-densitometric analysis of umbelliferone and psoralen in aegle marmelos. Pharm. Biol. 46(4): 266-272.

Ebadi, M. (2006). Pharmacodynamic Basis of Herbal Medicine, 2nd Edition. Boca Raton, FL.

Finkel, T. and Holbrook, N.J. (2000). Oxidants, oxidative stress and the biology of ageing. Nature. 408: 239-247.

Giugliano, D. Ceriello, A. and Paolisso, G. (1996). Oxidative stress and diabetic vascular complications. Diabetes Care. 19(3): 257-267.

Gorelik S. Ligumsky M. Kohen R. Kanner J. (2008). A novel function of red wine polyphenols in humans: prevention of absorption of cytotoxic lipid peroxidation products. FASEB J. 22(1): 41-46. doi: 10.1096/fj.07-9041com. Epub 2007 Aug 21. PMID: 17712060.

Góth, L. (2006). Reactive oxygen species, hydrogen peroxide, catalase and diabetes mellitus. Redox Rep. 11(6): 281-282.

Guo, L. and Davies, S.S. (2012). Bioactive aldehyde-modified phosphatidyl ethanolamines. Biochimie. 95(1): 74-78.

Haque, M.A. Chowdhury, R. Sikder, B. Mahmud, S. Hasib-Al-Mahfuj, Sarker, M.R. and Monjur-AlHossain, A.S.M. (2016). Antinociceptive, anti-inflammatory and central nervous system depressant activities of crude methanol extract and its dichloromethane, chloroform and aqueous fractions of *Scurrula parasitica* (Linn.) leaves. J. Pharmacogn. Phytochem. 5(5): 93-98.

Jakus, V. (2000). The role of free radicals, oxidative stress and antioxidant systems in diabetic vascular disease. Bratisl. Lek. Listy. 101(10): 541-551.

Kalaivanan, K. and Pugalendi, K.V. (2011). Antihyperglycemic effect of the alcoholic seed extract of *Swietenia macrophylla* on streptozotocin-diabetic rats. Pharmacogn. Res. 3(1): 67-71.

Kukić, J. Popović, V. Petrović, S., Mucaji, P., Ćirić, A., Stojković, D. and Soković, M. (2008). Antioxidant and antimicrobial activity of *Cynara cardunculus* extracts. Food chemistry. 107(2): 861-868.

Kumawat, M. Sharma, T.K. Singh, I. Singh, N. Ghalaut, V.S. Vardey, S.K. and Shankar, V. (2013). Antioxidant enzymes and lipid peroxidation in type 2 diabetes mellitus patients with and without nephropathy. N. Am. J. Med. Sci. 5(3): 213-219.

Laldingngheta, J. Lalnundanga, M. and Vabeiryureilai, M. (2020). Phytochemical analysis and isolation of flavonoid compound from methanol extract of the leaves of *Scurrula parasitica* L. Int. J. Pharm. Sci. Res. 12(9): 4927-4932.

Leong, L.P. and Shui, G. (2002). An investigation of antioxidant capacity of fruits in Singapore markets. Food Chem. *76*(1): 69-75.

Lin, Y.L. Cheng, C.Y. Lin, Y.P. Lau, Y.W. Juan, I.M. and Lin, J.K. (1998). Hypolipidemic effect of green tea leaves through induction of antioxidant and phase II enzymes including superoxide dismutase, catalase and glutathione S-transferase in rats. J. Agric. Food Chem. 46(5):1893-1899.

Mahajan, N. Joshi, P. Kondawar, M. Senthil Kumar, K.L. and Vaidhyalingam,V. (2013). Anti-nociceptive Potential of Scurrula parasitica. An Unexploited Parasitic Plant. Pharmacol. Rev. 3(1): 2230-9861.

Mahreen, R. Mohsin, M. Nasreen, Z. Siraj, M. and Ishaq, M. (2010). Significantly increased levels of serum malonaldehyde in type 2 diabetics with myocardial infarction. Int. J. Diabetes Dev. Ctries. 30(1): 49-51.

Mateos, R. Lecumberri, E. Ramos, S. Goya, L. and Bravo, L. (2005). Determination of malondialdehyde (MDA) by high-performance liquid chromatography in serum and liver as a biomarker for oxidative stress: Application to a rat model for hypercholesterolemia and evaluation of the effect of diets rich in phenolic antioxidants from fruits. J. Chromatogr. B Biomed. Appl. 827(1): 76-82.

Moussa, H.R. and Abdel-Aziz, S.M. (2008). Comparative response of drought tolerant and drought sensitive maize genotypes to water stress. Aust. J. Crop Sci. 1(1): 31-36.

Muhammad, K.J. Jamil, S. and Basar, N. (2019). Phytochemical study and biological activities of *Scurrula parasitica* L (Loranthaceae) leaves. J. Res. Pharm. 23(3): 522-531.

Okudu, T. Yoshida, T. and Hatano, T. (1994). Food phytochemicals for cancer prevention II. In Ho C. T, Osawa T, Huang MT, Rosen R.T. Chemistry and antioxidative effects of phenolic compounds from licorice, tea and Compositae and Labiateae herbs: Washington, DC: American Chemical Society. 132-143.

Proestos, C. Bakogiannis, A. Psarianos, C. Koutinas, A.A. Kanellaki, M. and Komaitis M. (2005). High performance liquid chromatography analysis of phenolic substances in Greek wines. Food Control. 16(4): 319-323.

Puneetha, G.K. and Amruthesh, K.N. (2016). Phytochemical screening and *in vitro* evaluation of antioxidant activity of various extracts of *Scurrula parasitica* L. Int. J. Pharm. Biol. Sci. 6(1): 77-86.

Saddala, R.R. Thopireddy, L. Ganapathi, N. and Kesireddy, S.R. (2013). Regulation of cardiac oxidative stress and lipid peroxidation in streptozotocin-induced diabetic rats treated with aqueous extract of *Pimpinella tirupatiensis* tuberous root. Exp. Toxicol. Pathol. 65(1-2): 15-19.

Sani, M.F. Kouhsari, S.M. and Moradabadi, L. (2012). Effects of three medicinal plants extracts in experimental diabetes: Antioxidant enzymes activities and plasma lipids profiles incomparison with metformin. Iran J. Pharm. Res. 11(3): 897-903.

Sardesai, V.M. (1995). Role of antioxidants in health maintenance. Nutr. Clin. Pract. 10: 19-25.

Shawki, H.A. Elzehery, R. Shahin, M. Abo-Hashem, E.M. and Youssef, M.M. (2021). Evaluation of some oxidative markers in diabetes and diabetic retinopathy. Diabetol. Int. 12(1): 108-117.

Singleton, V.L. and Rossi, J.A. (1965). Colorimetry of total phenolics with phosphomolybdic-phosphotungstic acid reagents. Am. J. Enol. Vitic. 16(3): 144-158.

Sundaram, R.K. Bhaskar, A. Vijayalingam, S. Viswanathan, M. Mohan, R. and Shanmugasundaram, K.R. (1996). Antioxidant status and lipid peroxidation in type II diabetes mellitus with and without complications. Clinical Science London, England: 1979. 90(4): 255-260.

Syyeda Anees, N.P. Siraj, M. and Ishaq, M. (2014). Evaluation of oxidative stress and antioxidant status in relation to glycemic control in Type 1 and Type 2 diabetes mellitus patients. Am J. Biochem Mol Biol. 4(2): 93-98.

Tiedge, M. Lortz, S. Drinkgern, J. and Lenzen, S. (1997). Relation between antioxidant enzyme gene expression and antioxidative defense status of insulin-producing cells. Diabetes 46(11): 1733-1742.

Waggiallah, H. and Alzohairy, M. (2011). The effect of oxidative stress on human red cells glutathione peroxidase, glutathione reductase level and prevalence of anemia among diabetics. N Am J. Med Sci. 3(7): 344-347.

West, I.C. (2000). Radicals and oxidative stress in diabetes. Diabet. Med. 17(3):171-180.

Yu, B.P. (1994). Cellular defenses against damage from reactive oxygen species. Physiological reviews. 74(1): 139-162.

Zakaria, Z.A. Somchit, M.N. Sulaiman, M.R. and Mat-Jais, A.M. (2004). Preliminary investigation in the antinociceptive properties of haruan (*Channa striatus*) fillet extracted with various solvent systems. Pak. J. Biol.Sci. 7(10): 1706-1710.

Zhu, Q., Qian, Y., Zong-Ping, Z.Z.L. Chen, F., Wang, M. (2013). Natural polyphenols alle*via*ted lipid peroxidation-induced modification on BSA. Journal of Functional Foods. 5(1): 355-361.