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Hyperlipidemia is one of the major health concerns worldwide. The present study aimed at utilizing clinicopathological tools to investigate the possible improving effect of *Avena sativa* mucilaginous extract (ASE) on lipid metabolic and liver function profiles in albino rats. The rats were rendered hyperlipidemic by 6-week supplementation of high-fat diet *ad libitum*. The rats were grouped into seven groups; with different treatment applications. Rats of group-I received normal diet and served as normal control; those of group-II were kept on high-fat (cholesterol 1% + coconut oil 2%) diet for 6 weeks and served as diseased control. Rats in group-III were kept on high-fat diet and received ezetimibe (1 mg/Kg B. Wt, orally, daily) and served as standard. Those in group-IV and V were kept on high-fat diet and received ASE at doses of 25 and 50 mg/Kg B. Wt., orally, daily (small and high doses, SD and HD, respectively) and served as treated-SD and treated-HD, respectively. While the last two groups (VI and VII) were kept on normal diet and received SD and HD of ASE. Blood samples for serum were taken for clinicochemical analysis on days 28 (4 weeks) and 42 (6 weeks) of the experiment and tissue specimens were taken for histopathology. ASE significantly (P<0.05) decreased the elevated serum lipid profile parameters, including total lipids, tri-acylglycerols (TAGs), cholesterol, LDL-C, VLDL-C, but significantly (P<0.05) normalized the serum HDL-C concentrations of rats kept on high-fat diet. Administration of *A. sativa* extract significantly decreased elevated serum liver enzyme activities in samples taken from animals kept on high-fat diet compared to the diseased untreated ones. Observations from histopathological examination were parallel and explanatory to clinicochemical analytical results. These data may suggest that the aqueous mucilaginous extract of *A. sativa* seed has a good health impact in cases associated with hyperlipidemia indicated by clinical pathology.

Key words: *Avena sativa*, antihyperlipidemic, clinical pathology, liver function, phytotherapy, phytomedicine.

INTRODUCTION

Among metabolic disorders, dyslipidemia, particularly hypercholesterolemia is considered as a major contributor in cardiovascular disease, including athero-sclerosis and atherosclerosis- associated conditions as coronary heart disease, ischemic cerebrovascular disease and peripheral vascular disease (Nelson, 2013). The best target for therapy in these conditions is to normalize blood and/or tissue cholesterol and lipid values. The conventional anti-hyperlipidemic chemical drugs are, usually, associated with some common adverse effects such as malaise,
gastric irritation, nausea/vomiting/diarrhea, hyperuricemia, muscle aches, red dry skin, and altered liver function; Kumar et al., 2008). Natural products with health improving impacts constitute, therefore, an interesting research area in minds of concerned personnel, including patients, physicians and researchers based on their safety, efficacy and easy access (Ververidis et al., 2007).

Avena sativa (Oat; Kingdom: Plantae; Family: Poaceae) and its constituents were reported to possess variable beneficial health activities like antimicrobial (Maizel et al., 1964), antiparkinsonian (Zhou and Panchuk-Voloshina, 1997), topical anti-inflammatory (Boyer et al., 1998; Capasso, 2003). Also included are wound healing (Aries et al., 1999), antiobesity (Jenkins et al., 2002), antioxidant (Bhatia, 2008), and anticancer (Anderson et al., 2009) activities.

Although effect of Avena sativa was tried on lipid profile in earlier studies (Karmally et al., 2005; Robitaille et al., 2005), yet, most of them used it as cereal as a whole or a part of the diet; and at least to our information, no studies have been performed using it as mucilaginous aqueous extract with adjusted doses. Moreover, Kerckhoffs et al. (2003) reported that the cholesterol lowering effect of β-glucan from oat bran in mildly hyper-cholesterolemic subjects may decrease when β-glucan is incorporated into bread and cookies. Therefore, the present study was designed to assess, using clinico-pathological tools, the hyperlipidemia improving profile of A. sativa mucilaginous aqueous extract as a natural remedy to the control of dyslipidemia in rats prepared as hyperlipidemic model by high-fat diet supplementation.

**MATERIALS AND METHODS**

**A. sativa mucilaginous extract**

The extract was obtained and standardized according to the classical methodological stepwise described by Harborne (1973) with minor modifications. The green seeds with aerial parts of A. sativa (Figure 1) were collected from the local environment (Qalubiubya Governorate, 2016) and identified by a plant specialist. Plant parts were refluxed in running tap water and then with bi-distilled water, shade dried at room temperature and chopped. Extract was prepared by macerating a weighed amount of the chopped plant parts (200 g) in a known volume (1.5 L) of water/ethanol (70:30, v/v). Maceration continued for 48 hours in refrigerator with occasional shaking. The hydro-ethanolic extract was then strained through muslin mesh and then concentrated using a shaking water bath at 56°C in a wide-mouthed containers and the mucilage obtained (yield) was then weighed and re-constituted freshly every day by dissolving in measured amount of bi-distilled water. Two stock solutions were prepared, 5 and 10 mg/ml. Avena sativa extract (ASE) was administered to rats at dosage rate of 25 (small dose; SD) and 50 (high dose; HD) mg/Kg B. Wt daily adjusted so that each rat (weighing 200 g) receives 1 ml of ASE orally using a rat gastric tube to the corresponding groups as explained below.

**Ezetimibe**

EZE, a standard inhibitor of cholesterol absorption, (Lipka, 2003) used in the present study was kindly gifted by SIGMA pharmaceuticals, Quesna Industrial Zone, Egypt. It was obtained as a pure powder. EZE was dissolved in 20% ethanol; where 10 mg of EZE were dissolved firstly in 10 ml absolute alcohol, and then the alcoholic solution of EZE was completed up to 50 ml by bi-distilled water. Each rat within the target group received 1 ml of the prepared solution which is equivalent to a dosage rate of 1 mg/Kg B. Wt.; orally, once daily (Patel, 2004).

**Experimental animals and protocol**

Forty-two white male rats, of age 6-8 weeks and of average weight of 200 g were used in a parallel study design. Rats were divided in separate cages and gained access to clean water and diets ad libitum at room temperature. A week later (for acclimatization), rats were subjected to various experimental treatments, as follows:

**Group-I**: Rats kept on normal balanced diet and administered no drugs but their vehicles; served as normal control.

**Group-II**: Rats kept on high-fat diet and administered no drugs but their vehicles; served as diseased control.

**Group-III**: Rats kept on high-fat diet and administered EZE at a dose rate of 1 mg/Kg B. Wt.; used as a standard anti-hyperlipidemic.

**Group-IV**: Rats kept on high-fat diet and administered ASE at a dose of 25 mg/Kg.

**Group-V**: Rats kept on high-fat diet and administered ASE at a dose of 50 mg/Kg.

**Group-VI**: Rats kept on normal balanced diet and administered ASE at a dose of 25 mg/Kg.

**Group-VII**: Rats kept on normal balanced diet and administered ASE at a dose of 50 mg/Kg.

Treatments were conducted simultaneously from the first day of the experiment and continued for 6 weeks with continuous observation. All procedures were ethical to animals, and were performed with merciful and humane manner under light ether anesthesia, and adhered to principles published by International Council for Laboratory Animal Science (ICLAS) and those of Benha University Animal Care and Use Committee.

**Sampling**

Blood for serum was collected on the 28th and the 42nd days from the start of the experiment. Blood was harvested into plain sampling tubes from the ocular medial canthus venous plexus using heparinized capillary tubes. The harvested blood was left to...
coagulate at 37°C for an hour; and then the clot was allowed to shrink by refrigeration for another hour. Centrifugation at 1000 × g for 10 min were performed to separate clean sera, which were collected into Eppendorf’s tubes using Pasteur pipettes. The obtained samples were at -50°C until analysis of the following clinicchemical parameters: Total lipids (TL), total cholesterol (TC), triacylglycerols (TAGs), high-density lipoprotein-cholesterol (HDL-C), low-density lipoprotein-cholesterol (LDL-C), very low-density lipoprotein-cholesterol (VLDL-C), aspartate amino transferase (AST), and alanine amino transferase (ALT). On the last day of the experiment, and after blood collection, animals were sacrificed by slaughtering using sharp scalpels under ether anesthesia, and tissue specimens from the liver were taken into 10% formalin solution for histopathological examination.

Clinicochemical analysis

The serum total lipids were determined according to the method described by Chabrol and Castellano (1961) using a kit supplied by Spinreact® (Sant Esteve De Bas, Spain); total cholesterol was determined enzymatically according to the method described by Meliattini et al. (1978) using a kit supplied by Spinreact®; HDL-L was determined according to the precipitation method described by Friedewald et al. (1972) using a kit supplied by Spectrum® (Obour city, Egypt); TAGs were measured enzymatically depending on the method explained after Young et al. (1975) using a kit Spinreact®. LDL-C and VLDL-C values were calculated using the formulae described by Friedewald et al. (1972) and Bauer (1982), respectively. Serum AST and ALT were quantitatively determined according to the method described by Murray (1984) using kits supplied by Diamond® (Cairo, Egypt). All laboratory steps were according to the instructions supplied by the kit manufacturers.

Histopathological examination

Liver specimens picked out from dissected rats in all groups were immediately placed in 10% formalin solution; processed for sectioning, staining and microscopical examining as described by Bancroft and Gamble (2008). Samples were allowed to fix for a 24 h period. The fixed samples were then gently washed under slowly running water for over-night. The clean fixed samples were then allowed to dehydrate in a series of increased-concentrations of ethanol starting in 70% and finalizing in absolute alcohol. The fixed, clean, dehydrated samples were placed in xylol for 3 h to clear and then placed in melted paraffin wax tissue boxes. The wax containing tissue specimens was left to solidify and then sections of 4 - 6 µm thickness were obtained using a rotary microtome. Staining procedure started with removal of wax from the microsections by two changes in ethanol (absolute; 5 minutes each). Ethanol was washed away with water. Sections were stained with Haematoxylin and Eosin (HandE) for 10 minutes, and then the extra stains were gently flushed under running water for 15 minutes. The stained microsections were dehydrated by alcohol series as mentioned above, then allowed to clear in xylol and finally covered with Dibutylphthalate Polystyrene Xylene (DBX). The prepared microsection slides were examined microscopically and interpreted by a specialist.

Data analysis and presentation

Data were presented as mean ± S.E of 6 observations. The obtained data were statistically analyzed using repeated-measures ANOVA at two timing points (4 and 6 weeks) with Tukey’s post-hoc to determine differences between groups at probability level of 5%. All statistical procedure was done by SPSS software, version 20. The sample size for each group was adjusted according to principles stated in literature, where value of α = 1.96, β = 0.842, a difference of 15 in each parameter is proposed between the groups as significant (Mean1− Mean2) and be detected with 80% beta power at significance level alpha of 0.05 (Das et al., 2016).

RESULTS

Effect of ASE on serum lipid profile:

As shown in Tables 1, 2, 3, 5, 6, there were significant
increases in serum total lipids, cholesterol, tri-acyl-
glycerols, LDL-C, VLDL-C values in samples obtained
from rats kept on high-fat diet, compared to those
obtained from rats on balanced diet. While administration
of ASE to control rats revealed insignificant alterations in
these parameters throughout the experimental period,
yet, its administration significantly (P<0.05) decreased
their serum levels in samples of rats kept on high-fat diet
in a dose-dependent manner. Nevertheless, as presented
in Table 4, there was a significant (P<0.05) decrease in
HDL-C values of samples of rats kept on high-fat diet.
Such decrease was significantly (P<0.05) not only
improved but also increased upon administration of ASE.
Again, there was no significant changes in rats kept on
basal diet.

**Effect of ASE on Liver function profile**

Data of the present study (Tables 7 and 8) demonstrate a
significant elevation in serum ALT and AST activities in
samples taken from rats kept on high-fat diet throughout
the experiment, compared to those in samples taken from
the normal control rats which were kept on balanced diet.
Although administration of ASE to normal rats revealed
insignificant alterations in liver enzyme activities
throughout the experimental period; yet, it significantly
(P<0.05) decreased their elevated serum activities in
animals kept on high-fat diet compared to the diseased
ones, upon its administration.

**Effect of ASE on Liver structure**

Data of the present study showed that there were no
changes in liver samples picked out from rats of normal
control group. Yet, subjecting experimental rats to high-
fat diet (diseased-control group) resulted in some hepatic
degenerative changes as hydropic and vacuolar
degenerations and peri-portal fatty change of hepatocytes,
as well as congestion of hepatic blood vessels. The severity and development of such changes
were inhibited largely, in liver specimens picked out from
ASE-treated groups and EZE-treated rats, as well (Figure
2a, b, c and d).

**DISCUSSION**

It is well established that alteration in lipid metabolic
profile, especially long standing hyperlipidemia is a direct
cause to various disease conditions including
atherosclerosis, ischemic heart disease (Ross and
Harker, 1976). This fact is later supported by Ross (1986)
and Crowther (2005). Findings of the current experiment
show that hyperlipidemia, induced by 42-day
supplementation of high-fat (cholesterol and coconut oil 1
and 2% wt/wt, respectively) diet caused, as expected,
marked alterations in the measured lipid parameters
(Table 1 to 6) of rat groups kept on such diet. In
addition, the established dyslipidemia was associated
with abnormally elevated liver function markers (AST and
ALT; Tables 7 and 8), together with considerable hepatic
histopathological changes (Figure 2). These rats were
applied, in the present study, as a model for
hyperlipidemia to evaluate the possible anti-
hyperlipidemic potential of a mucilage extract prepared
from *Avena sativa* green seeds and leaves.

Data presented in this study (Table 1) show a
significant (P<0.05) increase in serum total lipid value
of rats received high-fat diet throughout the 42 days of the
experiment, compared to the normal control rats fed on a
balanced diet. Similar findings were reported previously
in rats (Csont et al., 2002; El-Mahmoudy et al., 2013; El-
Mahmoudy et al., 2014) and rabbits (Diaz et al., 2000).

Administration of ASE significantly decreased serum
total lipid concentration in animals received high-fat diet
compared to the diseased untreated ones. The decrease
in serum total lipid concentration in ASE-treated animals
is logic after recording the improved serum values of
TAGs, TC, LDL-C and VLDL-C that were observed
simultaneously in this study. Lowering serum lipid profile
recorded in the present study may be parallel with that
reported by (Czerwiński et al., 2004) who found that oat
and amaranth meals positively affect plasma lipid profile
in rats fed cholesterol-containing diets. The authors
attributed such effect to the contents of the bioactive
components and the antioxidant activities of the studied
plant samples.

Findings presented in the current study (Table 2) show
a significant (P<0.05) elevation in serum TC
concentration in samples from rats kept on high-fat diet
throughout the 42-day experimental period, compared to

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Table 1. Serum total lipids concentration (mg/dL) in rats under different treatments, Normal Control (I), Hyperlipidemic (II), Hyperlipidemic with Ezetimibe 1 mg/Kg B. Wt., orally (III), Hyperlipidemic with ASE 25 and 50 mg ASE/Kg B. Wt. orally (IV and V), and Normal with ASE 25 and 50 mg ASE/Kg B. Wt. orally (VI and VII) daily for 42 days (X ± S.E; n=6).

<table>
<thead>
<tr>
<th>Time/Group</th>
<th>I</th>
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<th>VI</th>
<th>VII</th>
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<tr>
<td>Day 28</td>
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<tr>
<td></td>
<td>355.20±12.55</td>
<td>650.67±22.13</td>
<td>410.04±15.61</td>
<td>457.33±23.33</td>
<td>421.33±21.82</td>
<td>351.20±17.65</td>
<td>335.20±16.52</td>
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<tr>
<td>Day 42</td>
<td>362.15±19.25</td>
<td>714.61±25.33</td>
<td>430.25±25.33</td>
<td>475.66±24.84</td>
<td>443.00±22.67</td>
<td>345.15±15.85</td>
<td>332.15±16.66</td>
</tr>
</tbody>
</table>

Superscripts a and b mean significantly (P<0.05) different from corresponding Control (I) and Diseased (II), respectively.
that of the normal control animals. This finding is in accordance with that achieved by (Diaz et al., 2000) who found that rabbits kept on atherogenic diet exhibited marked elevation in TC in plasma.

Elevated serum cholesterol level is expected after high-fat diet supplementation and the inhibited clearing rate of LDL-C from the blood due to some defect in LDL receptors associated with elevated plasma total cholesterol values above normal levels (Zulet et al., 1999). Although administration of ASE to control rats revealed insignificant alteration in serum total cholesterol value throughout the experiment; yet, it significantly (P<0.05) decreased serum cholesterol concentration in animals received high-fat diet compared to the diseased untreated ones. The hypo-cholesterolemic effect of ASE may be explained based on decreasing cholesterol intestinal absorption with less dietary cholesterol is delivered to the pool of cholesterol to the liver; and/or greater clearance of LDL-cholesterol particles by the liver.

Findings presented in the current study (Table 3) show a significant (P<0.05) increase in TAGs in samples from animals kept on high-fat diet if compared with those of the normal control. Such significant elevation in serum TAGs may be explained on the basis of the diminished activity of lipase-insulin-dependent enzyme-contributing in TAGs clearance from blood by enhancing their hydrolysis to glycerol and free fatty acids (Yost et al., 1995). Daily oral administration of ASE significantly decreased serum TAGs concentration in animals received high-fat diet compared to that of diseased untreated ones. This finding may be parallel to those of Maier et al. (2000) and Czerwiński et al. (2004) in women and rats, respectively. The significant decrease in plasma TAGs has been explained previously by Bennani-Kabchi et al. (2000) who related that decrease to the higher rate of lipolysis mediated by enhanced plasma lipase activity. Nevertheless, more earlier, Griffin et al. (1982) stated that the lower level plasma TAGs might also reflect a lower rate of lipogenesis in the liver.

Tables 4, 5 and 6 present significant increases in serum LDL-C and VLDL-C and a significant decrease in HDL-C in rats kept on high-fat diet, compared to the corresponding normal control ones. The elevated serum
Table 2. Serum cholesterol concentration (mg/dL) in rats under different treatments, Normal Control (I), Hyperlipidemic (II), Hyperlipidemic with Ezetimibe 1 mg/Kg B. Wt., orally (III), Hyperlipidemic with ASE 25 and 50 mg ASE/Kg B. Wt. orally (IV and V), and Normal with ASE 25 and 50 mg ASE/Kg B. Wt. orally (VI and VII) daily for 42 days (X ± S.E; n=6).

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<th>Time/Group</th>
<th>I</th>
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<th>VI</th>
<th>VII</th>
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<tr>
<td>Day 28</td>
<td>73.17±6.20</td>
<td>151.18±8.06</td>
<td>93.33±8.40</td>
<td>107.33±7.66</td>
<td>98.22±7.85</td>
<td>70.17±6.10</td>
<td>68.37±7.20</td>
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<tr>
<td>Day 42</td>
<td>75.35±7.88</td>
<td>198.35±14.69</td>
<td>117.34±10.35</td>
<td>138.21±9.78</td>
<td>125.22±6.80</td>
<td>68.35±6.78</td>
<td>65.55±6.69</td>
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Superscripts a and b mean significantly (P<0.05) different from corresponding Control (I) and Diseased (II), respectively.

Table 3. Serum Triacylglycerols concentration (mg/dL) in rats under different treatments, Normal Control (I), Hyperlipidemic (II), Hyperlipidemic with Ezetimibe 1 mg/Kg B. Wt., orally (III), Hyperlipidemic with ASE 25 and 50 mg ASE/Kg B. Wt. orally (IV and V), and Normal with ASE 25 and 50 mg ASE/Kg B. Wt. orally (VI and VII) daily for 42 days (X ± S.E; n=6).

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<td>Day 28</td>
<td>92.75±8.94</td>
<td>186.35±17.33</td>
<td>116.66±24.53</td>
<td>140.55±14.70</td>
<td>125.98±10.06</td>
<td>90.75±8.96</td>
<td>85.75±7.94</td>
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<td>Day 42</td>
<td>98.2±11.33</td>
<td>195.66±19.24</td>
<td>124.16±10.48</td>
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<td>134.12±11.21</td>
<td>93.2±10.03</td>
<td>84.2±8.33</td>
</tr>
</tbody>
</table>

Superscripts a and b mean significantly (P<0.05) different from corresponding Control (I) and Diseased (II), respectively.

Table 4. Serum HDL-C concentration (mg/dL) in rats under different treatments, Normal Control (I), Hyperlipidemic (II), Hyperlipidemic with Ezetimibe 1 mg/Kg B. Wt., orally (III), Hyperlipidemic with ASE 25 and 50 mg ASE/Kg B. Wt. orally (IV and V), and Normal with ASE 25 and 50 mg ASE/Kg B. Wt. orally (VI and VII) daily for 42 days (X ± S.E; n=6).

<table>
<thead>
<tr>
<th>Time/Group</th>
<th>I</th>
<th>II</th>
<th>III</th>
<th>IV</th>
<th>V</th>
<th>VI</th>
<th>VII</th>
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<td>Day 28</td>
<td>38.85±3.35</td>
<td>21.72±1.67</td>
<td>30.46±1.49</td>
<td>23.89±3.48</td>
<td>25.53±1.87</td>
<td>39.85±3.35</td>
<td>41.85±3.35</td>
</tr>
<tr>
<td>Day 42</td>
<td>41.73±3.81</td>
<td>16.53±1.33</td>
<td>31.62±1.80</td>
<td>25.16±3.14</td>
<td>27.97±2.49</td>
<td>41.73±3.71</td>
<td>43.73±3.66</td>
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Superscripts a and b mean significantly (P<0.05) different from corresponding Control (I) and Diseased (II), respectively.

Table 5. Serum LDL-C concentration (mg/dL) in rats under different treatments, Normal Control (I), Hyperlipidemic (II), Hyperlipidemic with Ezetimibe 1 mg/Kg B. Wt., orally (III), Hyperlipidemic with ASE 25 and 50 mg ASE/Kg B. Wt. orally (IV and V), and Normal with ASE 25 and 50 mg ASE/Kg B. Wt. orally (VI and VII) daily for 42 days (X ± S.E; n=6).

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<tr>
<th>Time/Group</th>
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<th>IV</th>
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<th>VI</th>
<th>VII</th>
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<tbody>
<tr>
<td>Day 28</td>
<td>15.66±1.66</td>
<td>63.33±3.88</td>
<td>31.66±7.33</td>
<td>45.79±4.85</td>
<td>39.01±4.00</td>
<td>14.67±1.67</td>
<td>13.63±1.67</td>
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<tr>
<td>Day 42</td>
<td>16.02±1.33</td>
<td>75.12±8.82</td>
<td>35.91±9.05</td>
<td>48.10±4.54</td>
<td>42.89±6.45</td>
<td>15.32±1.33</td>
<td>14.32±1.33</td>
</tr>
</tbody>
</table>

Superscripts a and b mean significantly (P<0.05) different from corresponding Control (I) and Diseased (II), respectively.

LDL-C and VLDL-C seemed to occur upon over-production of LDL beyond the capacity of LDL-receptors expressed on hepatocyte cell membranes. In addition, the dietary fat and cholesterol may alter the serum lipoprotein pattern and increases the cholesterol content in VLDL (Mahley and Holcombe, 1977). ASE administration revealed significant decreases in serum LDL-C and VLDL-C concentrations with a significant increase in HDL-C when compared to those of the rats received high-fat diet. Such improving effect of ASE may be attributed to the decreased absorption of fats supplemented to rats and/or increased peripheral and hepatic breakdown of cholesterol esters from VLDL-C and LDL-C. The compositional change of HDL-C might be speculated due to a probable activation of Lechithin-cholesterol acyltransferase (LCAT) that is stimulated firstly by exogenous cholesterol.

Tables 7 and 8 present significant (P<0.05) elevations in ALT and AST activities in samples taken from rats kept on high-fat diet, if compared with those of the control. ASE administration significantly protected against elevations of these hepatic function biomarkers compared to the diseased untreated rats. This protecting effect may be explained on the bases of improved cholesterol hepatic metabolism as well as inhibiting its intestinal cholesterol absorption. Histopathological findings come supportive to the biochemical analysis, where the fatty degenerative changes observed in liver
Table 6. Serum VLDL-C concentration (mg/dL) in rats under different treatments, Normal Control (I), Hyperlipidemic (II), Hyperlipidemic with Ezetimibe 1 mg/Kg B. Wt., orally (III), Hyperlipidemic with ASE 25 and 50 mg ASE/Kg B. Wt. orally (IV and V), and Normal with ASE 25 and 50 mg ASE/Kg B. Wt. orally (VI and VII) daily for 42 days (X ± S.E; n=6).

<table>
<thead>
<tr>
<th>Time/Group</th>
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<td>26.6±1.70</td>
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<td>25.33±1.70</td>
</tr>
<tr>
<td>Day 42</td>
<td>25.66±2.77</td>
<td>46.24±2.20</td>
<td>33.1±1.64</td>
<td>39.12±1.67</td>
<td>36.31±1.58</td>
<td>24.67±1.33</td>
<td>21.67±1.77</td>
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</tbody>
</table>

Superscripts a and b mean significantly (P<0.05) different from corresponding Control (I) and Diseased (II), respectively.

Table 7. Serum ALT concentration (U/L) in rats under different treatments, Normal Control (I), Hyperlipidemic (II), Hyperlipidemic with Ezetimibe 1 mg/Kg B. Wt., orally (III), Hyperlipidemic with ASE 25 and 50 mg ASE/Kg B. Wt. orally (IV and V), and Normal with ASE 25 and 50 mg ASE/Kg B. Wt. orally (VI and VII) daily for 42 days (X ± S.E; n=6).

<table>
<thead>
<tr>
<th>Time/Group</th>
<th>I</th>
<th>II</th>
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<th>IV</th>
<th>V</th>
<th>VI</th>
<th>VII</th>
</tr>
</thead>
<tbody>
<tr>
<td>Day 28</td>
<td></td>
<td>24.32±3.19</td>
<td>65.12±1.25</td>
<td>29.53±3.04</td>
<td>36.78±1.25</td>
<td>34.13±1.00</td>
<td>23.76±1.33</td>
</tr>
<tr>
<td>Day 42</td>
<td>25.66±2.82</td>
<td>86.25±1.86</td>
<td>35.73±3.66</td>
<td>43.66±2.75</td>
<td>38.01±1.66</td>
<td>24.61±1.33</td>
<td>21.73±3.66</td>
</tr>
</tbody>
</table>

Superscripts a and b mean significantly (P<0.05) different from corresponding Control (I) and Diseased (II), respectively.

Table 8. Serum AST concentration (U/L) in rats under different treatments, Normal Control (I), Hyperlipidemic (II), Hyperlipidemic with Ezetimibe 1 mg/Kg B. Wt., orally (III), Hyperlipidemic with ASE 25 and 50 mg ASE/Kg B. Wt. orally (IV and V), and Normal with ASE 25 and 50 mg ASE/Kg B. Wt. orally (VI and VII) daily for 42 days (X ± S.E; n=6).

<table>
<thead>
<tr>
<th>Time/Group</th>
<th>I</th>
<th>II</th>
<th>III</th>
<th>IV</th>
<th>V</th>
<th>VI</th>
<th>VII</th>
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</thead>
<tbody>
<tr>
<td>Day 28</td>
<td></td>
<td>33.17±3.60</td>
<td>88.35±5.33</td>
<td>41.25±2.39</td>
<td>57.70±4.22</td>
<td>50.50±2.59</td>
<td>32.50±1.98</td>
</tr>
<tr>
<td>Day 42</td>
<td>36.33±4.31</td>
<td>97.50±7.68</td>
<td>52.66±4.09</td>
<td>62.12±3.90</td>
<td>58.25±3.39</td>
<td>33.51±1.98</td>
<td>32.66±2.09</td>
</tr>
</tbody>
</table>

Superscripts a and b mean significantly (P<0.05) different from corresponding Control (I) and Diseased (II), respectively.

specimens from rats kept on high-fat diet were almost not observed upon concurrent administration of ASE (Figure 2). The recorded beneficial effects of ASE may be related to the active pharmacological constituents present in the extract, including β-glucan, avenanthramides, flavonoids, flavonolignans, triterpenoid saponins, sterols, and tocols. In addition, the mucilaginous nature of the extract may impede lipid absorption from the intestines upon oral administration (Singh et al., 2013; Miraj and Kiani, 2016).

Conclusion

The present findings suggest that A. sativa mucilage extract may protect the liver and the body against development of hyperlipidemia, indicated by clinicopathological analysis. The extract, therefore, may have a good health impact in dyslipidemia and concurrent illnesses.

CONFLICT OF INTERESTS

The authors have not declared any conflict of interests.

REFERENCES


Assessment of antioxidant and antidiabetic properties of Picralima nitida seed extracts

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Received 24 September 2018; Accepted 22 October, 2018

The study investigated the antioxidant potential as well as the inhibitory potential of the seed extracts of Picralima nitida on α-amylase and α-glucosidase enzymes. The methanolic, aqueous and coconut water extracts were obtained using 70% methanol, distilled water and coconut water, respectively. Antioxidant properties were studied in vitro using DPPH (1, 1-diphenyl-2-picrylhydrazyl) radical scavenging, Ferric Reducing Antioxidant Power (FRAP), Total Antioxidant Capacity (TAC), Hydroxyl Radical Averting Capacity (HORAC), Total Phenol Content (TPC), and Total Flavonoid Content (TFC) assays. Different concentrations (0.1 - 0.5 mg/ml) of the extracts were subjected to α-amylase and α-glucosidase inhibitory assays using acarbose as standard. Absorbance was measured at 540 (for α-amylase) and 405 nm (α-glucosidase). The percentage of α-amylase and α-glucosidase inhibitory activity of the extracts and their IC₅₀ values were determined. The seed extracts of P. nitida showed significant antioxidant potential. The inhibitory activity of the extracts on α-amylase and α-glucosidase compared favourably with acarbose with the methanolic extract possessing the highest inhibitory activity. The methanolic extract also possessed the highest antioxidant capacity with the lowest IC₅₀ value among the extracts. The results indicate that the seed extracts of P. nitida possess significant antioxidant properties and are effective inhibitors of α-amylase and α-glucosidase enzymes.

Key words: Antioxidants, α-amylase, α-glucosidase, diabetes, phenolics, flavonoids, Picralima nitida.

INTRODUCTION

Cellular damage caused by free radicals is believed to play a central role in the aging and in disease progression (Sies et al., 1992). Antioxidants are the first line of defence against free radical damage, and are therefore important for maintaining optimum health and wellbeing. Imbalance between free radicals and antioxidants leads to oxidative stress which in turn results in the development of pathological conditions, one of which is diabetes. Studies have revealed the inference of oxidative stress in diabetes pathogenesis by lipid peroxidation, decreased vitamin C levels, impaired glutathione metabolism and alteration in enzymatic systems (Ullah et al., 2016).

Diabetes mellitus is a major cause of morbidity and mortality in the world. About 451 million people (aged 18-99 years) across the globe are reportedly suffering from diabetes (Cho et al., 2018). In 2015, diabetes was one of the leading causes of non-communicable diseases (NCD).
death, contributing 1.5 million deaths globally (Wang et al., 2015) and 321,100 deaths in the African region (IDF, 2015). More than half of diabetic patients in Africa live in the most populous countries in the region - Nigeria, South Africa, Ethiopia and the Democratic Republic of Congo (IDF, 2015). Consumption of energy-rich diet, obesity and increase in sedentary lifestyle has been attributed to the rise in the number of diabetic cases (Sheeahan, 2003).

Diabetes mellitus is classified into two namely type 1 and type 2. Type 1, commonly referred to as insulin dependent diabetes mellitus (IDDM) is caused by immunological destruction of pancreatic cells which results in insulin deficiency (Hudson et al., 2002) while type 2, also known as non-insulin dependent diabetes mellitus (NIDDM), results from insulin resistance, a condition which is caused by reduced sensitivity of target tissues to the metabolic effect of insulin (Neuser et al., 2005). Although various conventional therapies abound, over 80% of rural dwellers developing countries still depend on medicinal herbs (Brownlee, 2001). The side effects associated with the use of insulin and oral hypoglycemic agents have also led to an increase in the demand for alternative approaches to treat diabetes (Kahn et al., 2001). Hence, in modern days, huge attention has been directed towards recognition of plants with antidiabetic ability that may be used effectively for human consumption (Grover et al., 2002). A lot of plants have been screened for antidiabetic activity with promising results. Compounds responsible for antidiabetic activity in these plants include complex carbohydrates, alkaloids, glycopeptides, terpenoids, peptides, steroids, flavonoids, lipids, coumarines, sulphur compounds, and inorganic ions (Tchinda et al., 2008).

Picralima nitida is a species of the genus Picralima. It belongs to the hunterieae tribe of Apocynaceae family and is commonly called Osi-Igwe in Ibo and Abere in Yoruba (Duwiejua et al., 2002). In other parts of West Africa, the plant is called Gbe-Fondagne in Benin Republic, Adangme in Ghana, Abureebissi in Cote d’Ivoire and Susubalunyi in Sierra Leon (Kpodar et al., 2015). Picralima nitida is extensively distributed across West-Central Africa. The tree of P. nitida is under storey, reaching up to 4 to 5 m in height. Its trunk is about 5-60 m in diameter; cylindrical in shape and the wood is a pale yellow and hard (Okonta and Aguwa, 2007). The flowers are white (about 3 cm long) and they have ovoid fruits which become yellowish when mature. The leaves are broad (3-10 cm) and oblong (6-20 cm long) with tough tiny lateral nerves of about 14 to 24 pairs (Duwiejua et al., 2002).

P. nitida has diverse applications in West African traditional medicine. Various parts of the plant such as the leaves, seeds, stem, bark and roots are used by herbalists for the treatment of fever, hypertension, jaundice, gastro-intestinal disorders and malaria (Falodun et al., 2006). Preparations from different parts of the plant are employed as crude drug or crude herbal extract as remedy for various kinds of human diseases (Duwiejua et al., 2002). The seeds are widely used in West Africa especially in Nigeria, Cote d’Ivoire and Ghana as antipyretic, aphrodisiac agents, and for the treatment of malaria, pneumonia and other chest-conditions (Falodun et al., 2006). Herbalists have also made claims for the efficacy of the coconut water extract of P. nitida seeds in the treatment of many diseases, including diabetes (Adegoke and Oloyede, 2013). The presence of saponins, alkaloids, glycosides, steroids and tannins in the seeds of P. nitida has been reported (Sunmonu et al., 2014). It has been shown that seeds of the plant are rich in amino acids, vitamins A and E, as well as in mineral elements such as zinc, iron and manganese (Nwaogu, 2016). Investigations into the biological activity of the seed extracts also revealed their antimicrobial, larvicidal and hyperproteinenaemic potential (Nwabor et al., 2014; Adegoke and Oloyede, 2013). This study is therefore aimed at evaluating the antioxidant properties of the seed extracts of P. nitida and their in-vitro anti-diabetic potential through the inhibition of alpha-amylase and alpha-glucosidase.

**MATERIALS AND METHODS**

**Sample collection**

Dried seeds of P. nitida were purchased from Ibode Market, Molete, Ibadan. The plant was identified and authenticated at the Herbarium, Department of Botany, Obafemi Awolowo University, Ile-Ife, Nigeria and deposited with the voucher number FPI – 2112.

**Preparation of extracts**

The seeds of P. nitida were air-dried and ground into fine powder. The methanolic, aqueous and coconut water extracts were separately prepared by mixing 20 g of the powder with 500 ml each of 70% methanol, distilled water and coconut water respectively. The various mixtures were stored in appropriately labeled conical flasks at room temperature for 48 h. The mixtures were sieved using cheese cloth to obtain the supernatants. The supernatants were then concentrated to dryness using a rotary evaporator to obtain the crude extracts: methanolic extract (ME), aqueous extract (AE) and coconut water extract (CE).

**Determination of 1, 1-Diphenyl-2-Picrylhydrazyl hydrate (DPPH) radical scavenging activity**

The 1, 1-diphenyl-2-picrylhydrazyl hydrate (DPPH) radical scavenging activity was measured according to the method of Blois (1958). To 1 ml of different concentrations of the extract and the standard (ascorbic acid), 1 ml of 0.3 mM DPPH in methanol was added and allowed to react. The mixture was vortexed and kept in a dark chamber at room temperature for 30 min to allow for reaction. The absorbance was then measured at 517 nm against a DPPH control containing only 1 ml of methanol instead of the extract. The antioxidant activity was then calculated using this formula:

\[
\text{Percentage scavenging activity} = \frac{\text{Absorbance (control)} - \text{Absorbance (sample)}}{\text{Absorbance (control)}} \times 100
\]
The 50% inhibitory concentration (IC\textsubscript{50}) was obtained from a linear regression plot of percentage inhibition against concentration of the extract.

**Ferric reducing antioxidant power (FRAP) assay**

FRAP assay was carried out as described by Benzie and Strain (1999). FRAP reagent was obtained by mixing a 300 mmol/L acetate buffer (pH 3.6) with 2, 4, 6-tri-(2-pyridyl)-1, 3, 5-triazine and 20 mmol/L FeCl\textsubscript{3}. An aliquot (50 μl) of the extract at 5 mg/ml and 50 μl of standard solutions of ascorbic acid (20, 40, 60, 80, and 100 μg/ml) were added to 1 ml of FRAP reagent and thoroughly mixed. Absorbance was measured at 593 nm wavelength after 10 min against the blank which contained 50 μl of distilled water. All measurements were taken at room temperature with samples protected from direct sunlight. The reducing power was expressed as ascorbic acid equivalent (mg AAE/g extract) which is defined as the concentration of antioxidant that gives a ferric reducing ability equivalent to that of the ascorbic acid standard.

**Evaluation of total antioxidant capacity**

The total antioxidant assay was carried out based on the reduction of Mo (VI) to Mo (V) and the subsequent formation of a green phosphate/Mo (V) complex at acidic pH (Prieto et al. 1999). The extract (0.3 ml) was mixed with 3 ml of reagent solution (0.6 M sulphuric acid, 28 mM sodium phosphate and 4 mM ammonium molybdate). The tubes containing the reaction solutions were incubated at 95°C for 90 min. The solution was cooled to room temperature and absorbance was then measured at 695 nm against the blank containing 0.3 ml of methanol. The antioxidant activity was expressed as ascorbic acid equivalent (mg AAE/g extract) which served as positive control.

**Hydroxyl radical averting capacity (HORAC)**

The ability of the seed extracts of *P. nitida* to scavenge the hydroxyl radical generated by the Fenton reaction was measured according to the modified method of Chung et al. (1997). The reaction mixture containing 200 μl of 10 mM FeSO\textsubscript{4}, H2O, 200 μl of 10 mM EDTA and 200 μl of 10 mM 2-deoxyribose was added to 1.2 ml of 0.1 M phosphate buffer (pH 7.4) containing 200 μl of the extract. Thereafter, 200 μl of 10 mM H\textsubscript{2}O\textsubscript{2} was added to the mixture and incubated for 4 h at 37°C. After incubation, 1 ml of 28% Trichloroacetic acid (TCA) and 1 ml of 1% Thiobarbituric acid (TBA) were added and placed in a boiling water bath for 10 min. The resultant mixture was then allowed to cool to room temperature and absorbance was measured at 532 nm in a UV-VIS spectrophotometer.

\[
\text{Radical scavenging activity} = \frac{\text{Absorbance (control)} - \text{Absorbance (sample)}}{\text{Absorbance (control)}} \times 100
\]

**Determination of total phenol content**

The total phenol content of the extracts was determined using the Folin-Ciocalteu’s method of Singleton and Rossi (1995) as described by Gulcin et al. (2003). To 0.1 ml of 5 mg/ml of extract was added 0.9 ml of distilled water. 0.2 ml of 10 % Folin reagent was then added. The resulting mixture was vortexed. After 5 min, 1 ml of 7% Na\textsubscript{2}CO\textsubscript{3} solution was then added to the mixture. The solution was diluted to 2.5 ml with distilled water and then incubated for 90 min at room temperature. The absorbance at 750 nm was then read against the blank. The total phenol content of the extract was then calculated as shown in the equation below and expressed as mg gallic acid equivalent (GAE)/g fresh weight. Analysis was done in triplicates.

\[
C = \frac{c \times V}{m}
\]

where C = total content of phenol compound in gallic acid equivalent (GAE); c = concentration of gallic established from the calibration curve (mg/ml); V = volume of extract/fractions (ml) and m = weight of the crude extract obtained.

**Determination of total flavonoid content**

The estimation of the total flavonoid content of the plant extracts was based on the aluminium chloride colorimetric method according to the method of Zhilen et al. (1999) and as described by Miliauskas et al. (2004). To 0.2 ml of extract was added 0.4 ml of distilled water. This was followed by the addition of 0.1 ml of 5% (w/v) sodium nitrite. After 5 min, 0.1 ml of 10% (w/v) aluminum chloride and 0.2 ml of 4% sodium hydroxide solution were added and the volume made up to 2.5 ml with distilled water. The absorbance was measured against the blank at 500 nm. The total flavonoid content of the plant extract was calculated with the equation below and expressed as mg rutin equivalents per gram of the extract. Analysis was done in triplicates.

\[
X = \frac{q \times V}{w}
\]

where X = total content of flavonoid compound in rutin equivalent; q = concentration of rutin established from the standard curve; V = volume of extract (ml) and w = weight of the crude extract obtained.

**In vitro alpha-amylase inhibitory activity**

Alpha-amylase inhibitory activity of the different seed extracts (methanolic, aqueous and coconut water) of *P. nitida* was carried out according to the method of Miller (1959) with slight modifications. In a 96-well plate, reaction mixture containing 50 μl phosphatase buffer (100 mM, pH = 6.8), 10 μl α-amylase (2 U/ml), and 20 μl of varying concentrations of the extracts (0.1, 0.2, 0.3, 0.4, and 0.5 mg/ml) was pre-incubated at 37°C for 20 min. Then, 20 μl of 1% soluble starch (100 mM phosphatase buffer pH 6.8) was added as the substrate and incubated further at 37°C for 30 min; 100 μl of the DNS color reagent was then added and boiled for 10 min. The absorbance of the resulting mixture was measured at 540 nm using Multiplate Reader (Multiskka thermo scientific, version 1.00.40). Acarbose at various concentrations (0.1-0.5 mg/ml) was used as the positive control while blank was used as the negative control. Each experiment was performed in triplicates. The results were expressed as percentage inhibition, which was calculated using the formula,

\[
\text{Inhibitory activity} (%) = (1 - \frac{As}{Ac}) \times 100
\]

where As is the absorbance in the presence of test substance and Ac is the absorbance of control.

**In vitro alpha-glucosidase inhibitory activity**

Alpha-glucosidase inhibitory activity of the different seed extracts of...
Table 1. Quantification of total phenolics and total flavonoids in *P. nitida* seed extracts.

<table>
<thead>
<tr>
<th>Extract</th>
<th>TPC (mg GAE/g)</th>
<th>TFC (mg RE/g)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aqueous</td>
<td>435.87 ± 1.76</td>
<td>309.7 ± 4.73</td>
</tr>
<tr>
<td>Methanol</td>
<td>386.53 ± 2.67</td>
<td>338.3 ± 4.73</td>
</tr>
<tr>
<td>Coconut water</td>
<td>356.53 ± 4.67</td>
<td>345.5 ± 8.59</td>
</tr>
</tbody>
</table>

Data are expressed as mean ± SEM (n = 3); GAE = Gallic Acid Equivalent; TPC = Total Phenolic Content; RE = Rutin Equivalent; TFC = Total Flavonoid Content.

Table 2. DPPH radical scavenging activity of *P. nitida* Seed extracts.

<table>
<thead>
<tr>
<th>Conc. (µg/ml)</th>
<th>Ascorbic Acid</th>
<th>AE</th>
<th>ME</th>
<th>CE</th>
</tr>
</thead>
<tbody>
<tr>
<td>100</td>
<td>74.00 ± 1.20</td>
<td>14.24 ± 1.33</td>
<td>15.99 ± 1.90</td>
<td>14.28 ± 1.20</td>
</tr>
<tr>
<td>200</td>
<td>74.28 ± 1.65</td>
<td>20.83 ± 1.69</td>
<td>19.40 ± 1.66</td>
<td>14.56 ± 1.77</td>
</tr>
<tr>
<td>300</td>
<td>74.47 ± 2.12</td>
<td>25.07 ± 1.20</td>
<td>21.90 ± 0.50</td>
<td>15.68 ± 1.67</td>
</tr>
<tr>
<td>400</td>
<td>75.11 ± 1.33</td>
<td>29.40 ± 1.45</td>
<td>30.32 ± 2.44</td>
<td>16.13 ± 1.90</td>
</tr>
<tr>
<td>500</td>
<td>75.30 ± 0.76</td>
<td>37.79 ± 2.31</td>
<td>41.57 ± 2.00</td>
<td>16.96 ± 1.50</td>
</tr>
<tr>
<td>IC&lt;sub&gt;50&lt;/sub&gt;</td>
<td>37.94 ± 1.98</td>
<td>744.3 ± 1.40</td>
<td>688.89 ± 1.66</td>
<td>5080.4 ± 2.33</td>
</tr>
</tbody>
</table>

Percentage inhibition is relative to control without added inhibitor.

*P. nitida* was carried out according to the method reported by Ranila et al. (2010) with slight modifications. In a 96-well plate, reaction mixture containing 50 µl phosphate buffer (100 mM, pH = 6.8), 10 µl alpha-glucosidase (1 U/ml), and 20 µl of varying concentrations of extract (0.1, 0.2, 0.3, 0.4, and 0.5 mg/ml) was preincubated at 37°C for 15 min. Then, 20 µl P-NPG (5 mM) was added as a substrate and incubated further at 37°C for 20 min. The reaction was stopped by adding 50 µl NaCO<sub>3</sub> (0.1 M). The absorbance of the released p-nitrophenol was measured at 405 nm using Multiplate Reader. Acarbose at various concentrations (0.1-0.5 mg/ml) was included as the positive control while blank served as the negative control. Each experiment was performed in triplicates. The results were expressed as percentage inhibition, which was calculated using the formula:

\[
\text{Inhibitory activity} \% = \left(1 - \frac{A_S}{A_C}\right) \times 100
\]

where As is the absorbance in the presence of test substance and Ac is the absorbance of control.

Statistical analysis

All the measurements were done in triplicate and results are expressed in terms of mean ± standard error of mean. IC<sub>50</sub> values were calculated using GraphPad Prism 5 version 5.01 (Graph pad software, Inc., La Jolla, CA, USA) statistical software.

RESULTS AND DISCUSSION

Percentage yield of extracts

The percentage yields of the methanolic, aqueous and coconut water extracts were 14.90, 16.55 and 33.75% of the starting material respectively.

Total phenol and total flavonoid content

The results of total phenolic contents in *P. nitida* extracts are shown in Table 1. The aqueous extract contains the highest amount of phenols while coconut water extract has the lowest phenolic content. Coconut water extract was shown to have the highest flavonoid content while the aqueous extract contained the least amount of flavonoids.

DPPH radical scavenging activity of *P. nitida* seed extracts

The extracts were found to possess concentration dependent inhibitory activity against DPPH radical as shown in Table 2. The order of decreasing activity was methanol extract > aqueous extract > coconut water extract. DPPH free radical scavenging potential of all the extracts was found to be lower than that of the reference compound ascorbic acid.

Hydroxyl radical averting capacity

The order of decreasing activity was methanol > coconut water > aqueous extract as seen in Table 3. Hydroxyl free radical scavenging potential of all the extracts was
Table 3. Hydroxyl radical averting capacity of *P. nitida* seed extracts.

<table>
<thead>
<tr>
<th>Conc. (µg/ml)</th>
<th>Ascorbic Acid</th>
<th>AE</th>
<th>ME</th>
<th>CE</th>
</tr>
</thead>
<tbody>
<tr>
<td>100</td>
<td>68.42 ± 1.56</td>
<td>18.32 ± 0.88</td>
<td>40.35 ± 1.99</td>
<td>42.11 ± 1.50</td>
</tr>
<tr>
<td>200</td>
<td>69.40 ± 0.98</td>
<td>23.98 ± 1.65</td>
<td>44.44 ± 1.78</td>
<td>43.47 ± 1.20</td>
</tr>
<tr>
<td>300</td>
<td>70.57 ± 1.11</td>
<td>26.12 ± 2.10</td>
<td>46.20 ± 2.00</td>
<td>44.25 ± 0.73</td>
</tr>
<tr>
<td>400</td>
<td>71.15 ± 1.20</td>
<td>28.85 ± 1.76</td>
<td>50.49 ± 0.78</td>
<td>47.56 ± 1.90</td>
</tr>
<tr>
<td>IC&lt;sub&gt;50&lt;/sub&gt;</td>
<td>30.70 ± 1.96</td>
<td>829.03 ± 1.44</td>
<td>346.16 ± 2.13</td>
<td>495.96 ± 1.77</td>
</tr>
</tbody>
</table>

Percentage inhibition is relative to control without added inhibitor.

Table 4. Ferric reducing antioxidant power of the *P. nitida* Seed extracts.

<table>
<thead>
<tr>
<th>Extract</th>
<th>Ascorbic acid equivalent (mg AAE/g)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aqueous</td>
<td>16.41 ± 0.73</td>
</tr>
<tr>
<td>Methanol</td>
<td>21.49 ± 0.46</td>
</tr>
<tr>
<td>Coconut water</td>
<td>2.76 ± 0.20</td>
</tr>
</tbody>
</table>

Data are expressed as mean ± SEM (n = 3).

Table 5. Total antioxidant capacity of the *P. nitida* seed extracts.

<table>
<thead>
<tr>
<th>Extract</th>
<th>Ascorbic acid equivalent (mg AAE/g)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aqueous</td>
<td>28.4 ± 1.70</td>
</tr>
<tr>
<td>Methanol</td>
<td>22.7 ± 1.20</td>
</tr>
<tr>
<td>Coconut water</td>
<td>27.7 ± 1.20</td>
</tr>
</tbody>
</table>

Data are expressed as mean ± SEM (n = 3).

found to be lower than that of the reference compound ascorbic acid. The extracts possess concentration-dependent inhibitory activity against hydroxyl radical.

**Ferric reducing antioxidant power**

Table 4 shows the results of the FRAP assay. The methanolic extract had the highest FRAP value while the coconut water extract has the least FRAP value.

**Total antioxidant capacity**

The order of total antioxidant capacity as shown in Table 5 is aqueous extract > coconut water extract > methanol extract.

**Alpha-anylase inhibitory activity**

Table 6 shows the results alpha-amylase inhibitory activity of *P. nitida* seed extracts. The methanolic, aqueous and coconut water extracts of the seeds of *P. nitida* showed reasonable inhibitory activity against α-amylase when compared with acarbose.

**Alpha-glucosidase inhibitory activity**

The methanolic, aqueous and coconut water extracts of the seeds of *P. nitida* showed reasonable inhibitory activity against α-glucosidase when compared with acarbose. The methanolic extract exhibited the highest activity as shown in Table 7. Free radicals are harmful by-products produced during cellular metabolism, which could lead to oxidative damage in the body (Abidi and Ali, 1999). Antioxidants play a significant role in the body’s defence system against damage by free radicals. Several studies have described antioxidant compounds with radical scavenging activity present in herbs, fruits, vegetables and cereals extracts (Gray et al., 2002; Nuuttila et al., 2003; Hou et al., 2005).

Phenols are important plant constituents due to their free radical scavenging ability by virtue of their hydroxyl groups. Flavonoids are one of the most diverse and
results of the present study indicate that the seed extracts of *P. nitida* are rich in phenols and flavonoids which may be attributable to their high antioxidant activity. The leaves and root bark of the species that can react with every possible molecule in living organisms, especially with proteins, DNA, and lipids (Mohammed et al., 2009). Hydroxyl radicals can rapidly initiate lipid peroxidation process by extracting hydrogen atoms from unsaturated fatty acids (Aruoma, 1998). They are able to do this because of the aromatic hydroxylation at the ortho-position of their phenolic rings (Lipinski, 2011). The electron or proton donation capacities of *P. nitida* seed extracts were further confirmed by the Fenton reaction system in a concentration-dependent manner similar to their DPPH radical scavenging activity. Nwankwo et al. (2017) reported a significant free radical scavenging activity of the ethanolic extract of *P. nitida* seeds on malaria-induced albino mice. This may be attributable to their high phenol and flavonoid content.

DPPH scavenging activities involve hydrogen atoms transfer and electrons transfer (Saravanan and Parimelazhagun, 2014). The results of the present study indicate that the seed extracts of *P. nitida* may contain some hydrogen donor molecules which may help in the reduction of free radical production. However, all seed extracts exhibited lower scavenging properties than the reference compound ascorbic acid as seen in their IC₅₀ values in Table 2. The methanolic extract showed the highest DPPH radical scavenging activity among the seed extracts of *P. nitida*. Similar findings of the DPPH scavenging activity of the extracts of the root bark and leaf extracts of *P. nitida* have been reported (Erharuyi et al., 2014; Teguwa et al., 2013).

The hydroxyl radicals are extremely reactive oxygen species that can react with every possible molecule in living organisms, especially with proteins, DNA, and lipids (Mohammed et al., 2009). Hydroxyl radicals can rapidly initiate lipid peroxidation process by extracting hydrogen atoms from unsaturated fatty acids (Aruoma, 1998). They are able to do this because of the aromatic hydroxylation at the ortho-position of their phenolic rings (Lipinski, 2011). The electron or proton donation capacities of *P. nitida* seed extracts were further confirmed by the Fenton reaction system in a concentration-dependent manner similar to their DPPH radical scavenging activity. Nwankwo et al. (2017) reported a significant free radical scavenging activity of the ethanolic extract of *P. nitida* seeds on malaria-induced albino mice. This may be attributable to their high phenol and flavonoid content.

**Table 6. Alpha-amylase inhibitory activities of *P. nitida* seed extracts.**

<table>
<thead>
<tr>
<th>Conc. (µg/ml)</th>
<th>Acarbose</th>
<th>ME</th>
<th>CE</th>
</tr>
</thead>
<tbody>
<tr>
<td>100</td>
<td>50.265 ± 0.075</td>
<td>28.425 ± 1.005</td>
<td>27.425 ± 0.335</td>
</tr>
<tr>
<td>200</td>
<td>55.305 ± 0.275</td>
<td>31.940 ± 1.170</td>
<td>31.435 ± 0.335</td>
</tr>
<tr>
<td>300</td>
<td>58.645 ± 0.075</td>
<td>39.130 ± 0.670</td>
<td>33.275 ± 0.165</td>
</tr>
<tr>
<td>400</td>
<td>62.390 ± 0.130</td>
<td>44.820 ± 1.000</td>
<td>36.790 ± 1.000</td>
</tr>
<tr>
<td>500</td>
<td>65.460 ± 0.150</td>
<td>47.660 ± 0.500</td>
<td>52.175 ± 1.005</td>
</tr>
<tr>
<td>IC₅₀</td>
<td>99.756 ± 1.650</td>
<td>510.221 ± 3.810</td>
<td>502.023 ± 3.280</td>
</tr>
</tbody>
</table>

Data are expressed as mean ± SEM (n = 3); percentage inhibition is relative to control without added inhibitor.

**Table 7. Alpha-glucosidase inhibitory activities of *P. nitida* seed extracts.**

<table>
<thead>
<tr>
<th>Conc. (µg/ml)</th>
<th>Acarbose</th>
<th>ME</th>
<th>CE</th>
</tr>
</thead>
<tbody>
<tr>
<td>100</td>
<td>2.880 ± 0.110</td>
<td>6.595 ± 0.055</td>
<td>15.300 ± 0.011</td>
</tr>
<tr>
<td>200</td>
<td>4.600 ± 0.720</td>
<td>7.430 ± 0.110</td>
<td>15.690 ± 0.055</td>
</tr>
<tr>
<td>300</td>
<td>4.375 ± 0.055</td>
<td>8.980 ± 0.110</td>
<td>23.060 ± 0.110</td>
</tr>
<tr>
<td>400</td>
<td>5.430 ± 0.110</td>
<td>11.300 ± 0.050</td>
<td>24.340 ± 0.055</td>
</tr>
<tr>
<td>500</td>
<td>6.760 ± 0.110</td>
<td>12.585 ± 0.055</td>
<td>25.335 ± 0.055</td>
</tr>
<tr>
<td>IC₅₀ (mg/ml)</td>
<td>2.120 ± 0.570</td>
<td>1.350 ± 1.940</td>
<td>0.890 ± 0.1010</td>
</tr>
</tbody>
</table>

Data are expressed as mean ± SEM (n = 3); percentage inhibition is relative to control without added inhibitor.
content and FRAP assay which agrees with the findings of Zheng et al. (2001) and Rajurkar and Hande, (2011) who reported strong correlations between total phenol content and FRAP assay.

Total antioxidant activity of *P. nitida* seed extracts was evaluated by the phosphomolybdenum method (Prieto et al., 1999). This involves the transformation of Mo (VI) to Mo (V) by the antioxidant compound phosphomolybdenum complex with a maximal absorption at 695 nm. The results obtained from phosphomolybic acid assay are shown in Table 5 with the aqueous extract having the highest total antioxidant capacity. Polyphenolic compounds contribute significantly to the total antioxidant capacity of plants. A major goal in the treatment of diabetes mellitus is to maintain near normal blood glucose levels in both the fasting and postprandial state (Bailey, 2000). One therapeutic method used to decrease postprandial hyperglycemia is to suppress glucose production and/or absorption from the gastrointestinal tract through inhibition of either α-amylase or α-glucosidase enzymes (Cheng and Funtus, 2005; Kim et al., 2006; Matsui et al., 2007; Bhandari et al., 2008). Alpha amylase catalyzes polysaccharides (starch) into various oligosaccharides and disaccharides. Disaccharides produced by α-amylase are hydrolyzed further by α-glucosidase to yield glucose and other monosaccharides, which are readily absorbed in the small intestines (Shaw et al., 2010). Both animal studies (Okoli et al., 2010; Singh et al., 2001, Matsui et al., 2007) and clinical studies (Chiasson et al., 2002; Rhabasa-Lhoret and Chisason, 2004) have shown that inhibitors of both α-amylase and α-glucosidase can suppress glucose production and absorption from the small intestine. Currently, some inhibitors of α-amylase and α-glucosidase such as acarbose, phaseolamin and voglibose are being used to suppress postprandial glucose levels in diabetic patients (Kim et al., 2006).

The hypoglycemic effects of the coconut extract of the seeds of *P. nitida* in alloxan-induced diabetic rats have been reported (Ajaoo et al., 2009). Adegoke and Oloyede (2013) also reported that the coconut water extract of the leaves of the same plant significantly lowered blood glucose and protein levels in alloxan-induced diabetic rats. The results obtained from the present study indicate reasonable α-amylase inhibitory activity of the methanolic, aqueous and coconut water extracts of the seeds of *P. nitida*, comparing favourably with acarbose with the methanolic extract exhibiting the inhibitory highest activity among the other extracts (Table 5). Similar in vitro studies have attributed the α-amylase inhibitory activity of some plant extracts to the presence of tannins (Bhandari et al., 2008), flavonoids, polyphenols and their glycoside derivatives (Campos et al., 2003). The methanolic, aqueous and coconut water extracts of the seeds of *P. nitida* also showed reasonable α-glucosidase inhibitory activity in comparison with acarbose. Again, the methanolic extract was observed to be the most potent inhibitor of α-glucosidase among the seed extracts of *P. nitida*. The α-glucosidase inhibitory activity of some plant extracts to the presence of flavonoids, polyphenols as well as their glycoside derivatives (Casirola and Ferraris, 2006; Andrade-Cetto et al., 2008). The order of activity of the extracts (methanolic > aqueous > coconut) was observed to be the same for both α-amylase and α-glucosidase. The difference in activity can be explained by the different polarities of the solvents, which selectively extracted targetable bioactive compounds relating to the inhibition of carbohydrate metabolism from seeds of *P. nitida*. Extracts of leaves of *P. nitida* have been reported to inhibit alpha-amylase and alpha-glucosidase (Kazeem et al., 2013).

Conclusion

The results of these investigations indicate that the seeds of *P. nitida* possess significant antioxidant properties. The results also support the traditional of the use of *P. nitida* seed extracts in the treatment of diabetes (Aguwa et al., 2001).

CONFLICT OF INTERESTS

The authors have not declared any conflict of interests.

REFERENCES


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Utilization, cultivation practice and economic role of medicinal plants in Debre Markos Town, East Gojjam Zone, Amhara Region, Ethiopia

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Medicinal plants are useful for primary healthcare as a remedy for diseases and injury, while they are also used traditionally as food and beverages. Despite the wide role of medicinal plant, traditional utilization and management practices are not well documented in Debre Markos Town. Thus, this study was conducted to assess utilization and cultivation practice of the medicinal plants and to identify commonly used medicinal plants in Debre Markos Town. Data on medicinal plants production and utilization practice and its role were collected using field observation and semi-structured questioners. The study revealed that total of 55 medicinal plant species belonging to 35 families were used to treat various human diseases. Majority of medicinal plants species (80\%) were cultivated. 48\% of respondents have medicinal plants in their home garden. Leaf (13.3\%) and root parts (13.3\%) are widely in drug preparation. Widely used remedy preparation form (46.9\%) is liquid, made by boiling. Oral method of administration accounts (36.7\%) followed by dermal application (30\%). The practice of using medicinal plants in the local people has significant in economic and social sense, save cost and time. Thus, it enhances strong economic capacity of the people through creating healthy, physically and mentally capable people. In general, Debre Markos Town is rich in source of medicinal plants and use of traditional medicine is common. Most of available medicinal plants are found under threats in the study area, which is one of the main reasons for the degradation and destruction of habitats is a major cause of the loss of medicinal plant. Therefore, documentation medicinal plants provide important data.

**Key words:** Medicinal plants, cultivation, use.

**INTRODUCTION**

Medicinal plants are important for health care and remedy for diseases and injury. They are also used traditionally for foods and drinks (Yirga et al., 2011). Early humans acquired the knowledge on the utilization of plants for

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disease prevention and curative purposes through many years experience, careful observations and trial and error experiments (Sofowora, 1982; Martin, 1985)

According to Bekele (2007), the major reasons why medicinal plants are demanded in Ethiopia are due to culturally linked traditions, the trust the communities have in traditional medicine, and relatively low cost in using them.

According to Bekele (2007) in Ethiopia, except in a few cases where a few food crops with medicinal value are cultivated, there is no organized cultivation of plants species for medicinal purposes. Systematic cultivation and conservation of medicinal plants requires the effort of all stakeholders including the traditional healers, researchers, academicians, farmers and the community at large. Incorporation of medicinal plants in agricultural fields and home gardens will help to increase supply and conservation of important medicinal plants (Oladele et al., 2011). There is little data available for any of medicinal plants for large and small-scale farming. Different studies have been made in different parts of the country, most of them are more general and do not focus on a specific agro-ecology of the country.

Aasmamaw and Achamyele (2018) assessed available medicinal plants in Gozamen Woreda Dalgaw kebele and found 37 for human disease treatment. Nigussie et al. (2018) also conduct assessment on medicinal plants in Gozamen Woreda. However, there is limited study on use and cultivation practice of medicinal plants in Debre Markos Town. Thus, this study was conducted to assess utilization and cultivation practice of the medicinal plants in Debre Markos Town. It is also important to identify commonly used medicinal plants and to document utilization practice of medicinal plants in Debre Markos Town.

MATERIALS AND METHODS

Description of the study area

Debre Markos town was the historical center of administration and commerce of Gojam for a long period of time. It is located 300 km Northwest of Addis Ababa and 265 km southeast of the Amhara National Regional State Capital City-Bahir Dar. The geographical coordinates of the town are 10°20’N 37°43’E. Its total municipal area is about 60 km square. It is situated at 2450 m above sea level. The weather condition, in most of the time is, ‘Woina Degä’. The town receives a mean annual rainfall of 1300-1380 mm and the temperature ranges between 15 and 22°C. According to the 2007 national survey, the total population of Debre Markos Town was 62,469 with a total of 18,479 households. The town has seven kebeles. The study was conducted in two kebeles (1 and 3).

Method

The study populations are the households who are living in the two kebeles of Debre Markos Town. Those persons who owned the houses by rent are not included in the study. Individuals aged greater than 18 years and living for at least six months in the town were involved in the study. Two kebeles were randomly selected from the seven kebeles. Accordingly, 30 informants were purposively selected from each kebele of total of 60 respondents. The household mothers and household head/husbands were asked about the cultivation and utilization practice of medicinal plants. Informants below 18 years are not believed to have enough indigenous knowledge.

Data were collected by both qualitative and quantitative methods. Data for the study was collected from both primary and secondary source of data. The primary data sources used were field visit, key informant interview and semi-structured questionnaires. The informants were convinced upon the purpose of the research and each informant was requested for permission before the interview. The interview prepared in English was translated into Amharic, the local languages of the informants. After researchers obtained an oral consent from each informant, information concerning the medicinal plant utilizers was collected. Medicinal plants information such as the plant local name, treated disease, and the use plant parts, preparation and route of administration etc. were recorded from the informants. The collected data was analyzed using SPSS software. We employed descriptive statistics and further inferential statistics to predict and indicate the utilization and cultivation practice of medicinal plants in the investigation area.

Ethical consideration

A formal letter was written from Department of Horticulture, College of Agriculture and Natural Resources, Debre Markos University, to get permission for conducting the research in the community. A verbal informed consent was taken from each household owner participants after clearly stating the purpose of the study.

RESULTS AND DISCUSSION

Demography

Regarding educational status of informants, from 60 respondents 10 respondents (16.7 %) are attend college level and above, 18 respondents (30 %) are illiterates, 2 respondents (3.3 %) are on primary school and 30 respondents (50 %) are able to read and write, and when we compare their sex 22 respondents are males. From the total population 14 persons (23.3 %) are government employed, 24 persons (40 %) are merchants, and 22 persons (36.7 %) are Unemployed Table 1.

Diversity of medicinal plants in the study area

A total of 54 medicinal plants (Table 2) were reported by respondents in the study area to treat different human ailments. This plant belongs to 35 families. The largest diversity species are Lamiaceae with 6 (10.91%) species, Solanaceae with 5 (90.9%) species and Asteraceae 4 (7.27%) species. The remaining 4 families had (3.64 %)
species each and 29 Families had one species each. This result indicates that the study area had widely consists diversity of the plant species found in families Lamiaceae, Solanaceae and Asteraceae. Similar results were reported by Adera (2014) who reported family Asteraceae was represented by 5 species followed by 4 species of Lamiaceae in Ghibmi district. Alemayehu et al. (2015) also reported that Lamiaceae and Solanaceae families was the widely distributed in Minjar Shenkora district. The study of Banjaw et al. (2016) at Wondogent area indicated that Lamiaceae family hold greater number of species followed by Asteraceae family. Giday et al. (2007) also reported similar results.

The present result also showed that peoples in the study area were widely used and provide priority for Medicinal Plants to treat human diseases like mich (fiver illness), Cough, Wound, Stomachache, Diarrhea, Evil eye, Snakebite, Throat infection, etc. The major reason of community to widely depend on medicinal plants is due to unaffordable price of modern medicine. The result is in line with those of Bekele (2007) that reported that the current demands for herbal remedies in both developed and developing countries are increasing. In developed countries, this may be partly due to the dissatisfaction with conventional medicines while with the developing countries this is due to lack of medical doctors, shortage of pharmaceutical products and their unaffordable prices. In the current study, one ailment can be treated with combination of plant species or single plant species (Table 5). Similar results were reported by Nigussie et al. (2018). The study of Zewdu (2013) at Gonder Zuria District, indicated 42 medicinal plant species representing 41 genera and 31 families to treat diseases (the highest number of uses mentioned for any disease were general health (69), respiratory (51), and gastrointestinal (28). According to Zerabruk and Yirga (2011), a total of 26 species of medicinal plants were collected and identified to treat 36 human ailments at Gindberet district, Western

### Table 1. Information about respondents.

<table>
<thead>
<tr>
<th>Questioners</th>
<th>Options</th>
<th>Frequency</th>
<th>Percentage (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Educational Status</td>
<td>College and Above</td>
<td>10</td>
<td>16.7</td>
</tr>
<tr>
<td></td>
<td>Illiterate</td>
<td>18</td>
<td>30.0</td>
</tr>
<tr>
<td></td>
<td>Primary school</td>
<td>2</td>
<td>3.3</td>
</tr>
<tr>
<td></td>
<td>Reading and writing</td>
<td>30</td>
<td>50.0</td>
</tr>
</tbody>
</table>

### Table 2. Taxonomic diversity of the medicinal plants in the study area.

<table>
<thead>
<tr>
<th>S/N</th>
<th>Family name</th>
<th>Number of species</th>
<th>Percentage (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Lamiaceae</td>
<td>6</td>
<td>10.91</td>
</tr>
<tr>
<td>2</td>
<td>Rutaceae</td>
<td>2</td>
<td>3.64</td>
</tr>
<tr>
<td>3</td>
<td>Brassicaceae</td>
<td>3</td>
<td>5.45</td>
</tr>
<tr>
<td>4</td>
<td>Solanaceae</td>
<td>5</td>
<td>9.09</td>
</tr>
<tr>
<td>5</td>
<td>Asteraceae</td>
<td>4</td>
<td>7.27</td>
</tr>
<tr>
<td>6</td>
<td>Rosaceae</td>
<td>2</td>
<td>3.64</td>
</tr>
<tr>
<td>7</td>
<td>Apiaceae</td>
<td>2</td>
<td>3.64</td>
</tr>
<tr>
<td>8</td>
<td>Euphorbiaceae</td>
<td>2</td>
<td>3.64</td>
</tr>
<tr>
<td>9</td>
<td>Other 29 families</td>
<td>29</td>
<td>61.72</td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td>55</td>
<td>100</td>
</tr>
</tbody>
</table>
Ethiopia.

Source of medicinal plants in the study area

The present study revealed that 80, 10, 4, 3 and 3% of respondents explained that medicinal plants for their use was obtained from cultivation in home garden, purchased from market, Traditional healer, neighbors and collected from wild habitat respectively (Figure 1). The present finding is in line with the findings of Feyyesa et al. (2015) who reported that most medicinal plants were obtained from cultivation in Jimma zone and Giday et al. (2007) who reported that major sources of medicinal plants in Agew Awi zone are home gardens or cultivation. According to WHO et al. (1993), the best way to provide the plant material needed for medicine is to cultivate the plants. This is far better than collecting the plant material from the wild since it does not deplete wild stocks, and in many cases, the declining habitats of native plants can no longer supply the expanding market for medicinal plant products. In the case of rare, endangered or over-exploited plants, cultivation is the only way to provide material without further endangering the survival of those species. Cultivation also has pharmacological advantages over wild-collection. Wild-collected plants normally vary in quality and composition, due to environmental and genetic differences. In cultivation, this variation - and the resulting uncertainty of the therapeutic benefit - is much reduced. The plants can be grown in areas of similar climate and soil, they can be irrigated to increase yields and they can be harvested at the right time. Cultivation also greatly reduces the possibility of mis-identification and adulteration.

Availability, management practice and utilization of medicinal plants

The present study indicates that 48% of respondents have medicinal plants in their home garden. Among these respondents, 33.4% respondents have four types of medicinal plants in their home garden, while 12 respondents have not cultivated medicinal plants and obtained from other sources. On the other hand, 44 respondents explained that they were given care and special management practices (irrigation, cultivation, fertilization and weeding) for medicinal plants whereas the remaining 4 respondents were does that did not give any care, but all respondents used medicinal plants for treatment of many human diseases (Table 3).

Plant parts used

The present finding revealed that in the study area, different plant parts were harvested (for example leaves, roots, seeds, stem and fruit) separately and used by mixing each other for preparation of traditional drugs. In the study area, the informants reported that 13.3% species of medicinal plants were harvested to use their leaves (13.3%) roots and (13.3%) by combining leaves and roots together in drug preparation (Figure 2). In the study area, also respondents explained that 6.7% used seed to treat diseases. Leaves are widely used plant parts for drug preparations than the other parts either individually or by mixing other plant parts. The present result is in line with the finding of Giday, (2001); Amenu (2007); Alemayehu et al. (2015); Banjaw et al. (2016); Asmamaw and Achamyeleh (2018). Harvesting leaves are common practice in the study area which results in a threat to rare plants. Although, the equivalent ratio of harvested part in study area was root separately and in combination with leaves; which negatively affects the growth and physiology of the plant results in the destruction of mother plant. Utilization of leaves for drug preparation is important for conservation of medicinal plants since harvesting leaves may not cause detrimental effect on the plants compared to the root or whole plant collections (Megersa et al., 2013). According to Hunde (2006) utilization of roots and whole plants may have negative consequences on the sustainability of the medicinal plant species in the area.

Form used

The major forms of preparation of plant medicines in the

<table>
<thead>
<tr>
<th>Questioners</th>
<th>Number</th>
<th>Frequency (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Presence of medicinal plants</td>
<td>60</td>
<td>48  12</td>
</tr>
<tr>
<td>Care given to medicinal plants</td>
<td>60</td>
<td>44  16</td>
</tr>
<tr>
<td>Use of medicinal plants</td>
<td>60</td>
<td>60  0</td>
</tr>
</tbody>
</table>

Table 3. Availability, management practice and utilization of medicinal plants.
The study area (46.9%) included liquid obtained after crushing and chopping forms by means of boiling, and absorbing the vapor part and drinking like water. 26.7% of respondents explain that they use medicinal plants in
solid form by inhaling in nasal method. 13.3% respondents used medicinal plants in liquid and gas form in combination (Figure 3). The popularity of the liquid preparations may be due to the easy method of the liquid preparation form and to the property and availability of water as solvent. The common utilization of the liquid preparations forms in the study area is in agreement with findings in other parts of the country reported by Abiyot (2002); Alemayehu et al. (2015) and Asmamaw and Achamyeleh (2018). Utilization of fresh materials of the plants species is more preferable than dried form. This is because fresh materials are harvested directly and used early before deterioration. Nevertheless, communities believe that fresh materials are effective in the treatment as the contents are not lost. This agrees with the findings of Tamene (2000) and Hunde (2001).

Preparation methods

The majority of the medicines (53.3%) in the study area are prepared through boiling only followed by boiling and chewing together (13.3%). Ten percent of respondents explained that they prepare medicinal plants in the form of smoking / fumigation (Figure 4). The present finding is in line with those of Assegid and Tesfaye (2014) and Birhanu and Ayalew (2018). During the preparations of the remedies, extracted medicines were mixed with honey, milk, water, coffee and tea which might be used to reverse adverse effect of the traditional medicines such as vomiting, itching and diarrhea. The same result was also reported by Assegid and Tesfaye (2014).

Administration method

Peoples in the study area mostly administer traditional medicine orally (Table 4). Oral method of administration only accounts for 36.7% followed by dermal application (30%) and dermal and nasal combination (20%) whereas the least used routes were nasal (10%). These results were similar to the findings of Hunde (2001), Giday (2001), Giday et al. (2007), Amenu (2007), Birhane et al. (2011), Assegid and Tesfaye, (2014), Alemayehu et al. (2015), Asmamaw and Achamyeleh (2018), and Birhanu and Ayalew (2018) who noted that drinking (oral application) was the dominant method of administration. Nigussie et al. (2018) conducted research in Gozamen District and reported that most common route of administration is internal particularly oral that accounted...
for 51.61% followed by dermal (24.73%). Dominant routes of administration are oral and dermal because they perceive rapid physiological reaction of the prepared medicines with the pathogens and increase its curative power. The route of administration of herbal medicines could be related to bioactive agents in the extracts of the plants (Gurib, 2006). For example, herbal medicines whose bioactive agents are alkaloids are easily assimilated when administered orally while terpenoids especially essential oils are best administered through dermal and/nasal routes (Boadu and Asase, 2017).

**Economic role of medicinal plants**

The practice of using medicinal plants by the local people has enormous and has paramount significance in economic and social sense. While practicing cultivating medicinal plants, they feel confident that they will cure different diseases by their own timely (Table 5). Especially, there are different infectious diseases and accidental illness that cause psychological and physical damage to the people and are being treated by those medical plants available in their surroundings. Moreover, those people who used medicinal plants for the sake of their health can save and reduce frequency of unnecessary waste from transportation and other miscellaneous cost to modern health institutions. Thus, it enhances strong economic capacity of the people through creating healthy, physically and mentally capable people. Furthermore, pre and post treatment practice that cure the disease in the traditional and routine practice in

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**Figure 4.** Methods to prepare remedies for medicinal plants.

**Table 4. Route of administration.**

<table>
<thead>
<tr>
<th>Route</th>
<th>Frequency</th>
<th>Percentage (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dermal</td>
<td>18</td>
<td>30</td>
</tr>
<tr>
<td>Oral</td>
<td>26</td>
<td>36.7</td>
</tr>
<tr>
<td>Dermal and Nasal</td>
<td>12</td>
<td>20</td>
</tr>
<tr>
<td>Dermal, Nasal and Oral</td>
<td>8</td>
<td>13.3</td>
</tr>
<tr>
<td>Nasal</td>
<td>6</td>
<td>10</td>
</tr>
<tr>
<td>Total</td>
<td>60</td>
<td>100</td>
</tr>
</tbody>
</table>
Table 5. Role and Utilization method of medicinal plants for the treatment of human diseases.

<table>
<thead>
<tr>
<th>S/N</th>
<th>Scientific name</th>
<th>Family</th>
<th>Local name/ Amharic</th>
<th>Disease Treated</th>
<th>Part used</th>
<th>Method of Preparation</th>
<th>Rout of Administration</th>
<th>Source of the plant</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Achyranthes aspera</td>
<td>Amaranthaceae</td>
<td>Telegn</td>
<td>Wound and skin cut</td>
<td>Leaf</td>
<td>Fresh leaves are crushed and then mashed on infected part</td>
<td>Dermal</td>
<td>Traditional healer</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Body swelling</td>
<td>Leaf</td>
<td>Leaves are powdered, mixed with butter and then mash on infected part</td>
<td>Dermal</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>34Excess male</td>
<td>Root</td>
<td>The root of is crushed and tie on abdominal part the body</td>
<td>Dermal</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>menstruation</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>and retained</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>placenta</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>Antidysentrica</td>
<td>Simaroubaceae</td>
<td>Avalo</td>
<td>Wound</td>
<td>Leaf</td>
<td>Chopped fresh leaves are mashed on wound mostly in child’s head</td>
<td>Dermal</td>
<td>Wild</td>
</tr>
<tr>
<td>3</td>
<td>Allium sativum L.</td>
<td>Alliaceae</td>
<td>Nech Shinkurit</td>
<td>Stomachache</td>
<td>Bulb</td>
<td>Its bulb mixed with seed of Lepidium sativum are crushed together and eaten with injera.</td>
<td>Oral</td>
<td>Market</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Asthma</td>
<td>Bulb</td>
<td>Fresh bulbs are chopped mixed with honey and eaten</td>
<td>Oral</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Influenza</td>
<td>Bulb</td>
<td>Chewing fresh bulb</td>
<td>Oral</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>Allium cepa</td>
<td>Alliaceae</td>
<td>Keyshinkurit</td>
<td>Cough</td>
<td>Bulb</td>
<td>Raw bulb is chopped and eaten with enjera</td>
<td>Oral</td>
<td>Market</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Hypertension</td>
<td>Bulb</td>
<td>Bulbs are crushed and immersed in little water drunk</td>
<td>Oral</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>Aloe vera</td>
<td>Alliaceae</td>
<td>Eret</td>
<td>Discourage the baby from Breast suckling</td>
<td>Leaf</td>
<td>Juice of the plant is creamed on the breast</td>
<td>Dermal</td>
<td>Home Garden</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Childbirth</td>
<td>Leaf</td>
<td>juice of the leaf is given to a mother to ease labour</td>
<td>Dermal</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Cool burns</td>
<td>Leaf</td>
<td>Fresh juice of the plant is smashed on infected part</td>
<td>Dermal</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>Artemisia abyssinica</td>
<td>Asteraceae</td>
<td>Anti/chikugn</td>
<td>Intestinal problems</td>
<td>Leaf</td>
<td>Fresh leaves are chopped mixed with water and drank</td>
<td>Oral</td>
<td>Home garden</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Evil eye</td>
<td>Leaf</td>
<td>Fresh leaves are chopped and smell</td>
<td>Nasal</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Common cold</td>
<td>Leaf</td>
<td>Fresh leaves are chopped and sniffed</td>
<td>Nasal</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Fibril Illness</td>
<td>Root</td>
<td>The root is crushed and drunk</td>
<td>Oral</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>Brassica nigra (L.)</td>
<td>(Brassicaceae)</td>
<td>Sinafich</td>
<td>Abdominal pain</td>
<td>Seed</td>
<td>Powder of dried seeds with seeds of Lepidium sativum mixed with water and drunk</td>
<td>Oral</td>
<td>Market</td>
</tr>
<tr>
<td>8</td>
<td>Capsicum annuum L.</td>
<td>Solanaceae</td>
<td>Kariya</td>
<td>Nausia</td>
<td>Fruit</td>
<td>Its fruits with garlic, ginger, and black cumin are immersed in water and drunk</td>
<td>Oral</td>
<td>Market</td>
</tr>
<tr>
<td>9</td>
<td>Carica papaya L.</td>
<td>Caricaceae</td>
<td>Papaya</td>
<td>Anaemia</td>
<td>Fruit</td>
<td>Extracted juice is mixed with sugar, and drunk in the morning</td>
<td>Oral</td>
<td>Market</td>
</tr>
<tr>
<td>10</td>
<td>Citrus limon</td>
<td>Rutaceae</td>
<td>Lomi</td>
<td>Stomach ache</td>
<td>Fruit</td>
<td>Its juice added to tea and honey together and drunk</td>
<td>Oral</td>
<td>Market</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Skin rash</td>
<td>Fruit</td>
<td>Its juice is mashed on the infected body</td>
<td>Dermal</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Nasal bleeding</td>
<td>Fruit</td>
<td>Its juice is added to nose or drank it</td>
<td>Nasal/oral</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Athletes foot</td>
<td>Fruit</td>
<td>Extracted juice is mashed on the leg</td>
<td>Dermal</td>
<td></td>
</tr>
</tbody>
</table>
Table 5. Contd.

<table>
<thead>
<tr>
<th>No.</th>
<th>Plant Name</th>
<th>Family</th>
<th>Part Used</th>
<th>Use</th>
<th>Route</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>11</td>
<td>Clerodendrum myricoides</td>
<td>Lamiaceae</td>
<td>Leaf</td>
<td>Wounds and Fire burn</td>
<td>Dermal</td>
<td>Traditional Healer</td>
</tr>
<tr>
<td>12</td>
<td>Coffea arabica</td>
<td>Rubiaceae</td>
<td>Seed</td>
<td>The powder is boiled and mixed with droplet of oil and drunk</td>
<td>Oral</td>
<td>Market</td>
</tr>
<tr>
<td>13</td>
<td>Coriandrum sativum</td>
<td>Umbelliferae</td>
<td>Seed</td>
<td>Seeds are roasted and boiled in water and drunk</td>
<td>Oral</td>
<td>Market</td>
</tr>
<tr>
<td>14</td>
<td>Croton acrostachyus</td>
<td>Euphorbiacea</td>
<td>Leaf</td>
<td>Fresh leaf or shoot juice is mashed on infected body</td>
<td>Dermal</td>
<td>Home Garden</td>
</tr>
<tr>
<td>15</td>
<td>Datura stramonium</td>
<td>Solanaceae</td>
<td>Leaf</td>
<td>The leaves are crushed, mixed with little water and 1 added few amount through ear</td>
<td>Ear</td>
<td>Wild</td>
</tr>
<tr>
<td>16</td>
<td>Dodonaea angustifolia</td>
<td>Sapindaceae</td>
<td>Leaf</td>
<td>Fresh leaf Juice is drunk</td>
<td>Oral</td>
<td>Wild</td>
</tr>
<tr>
<td>17</td>
<td>Echinops kebericho</td>
<td>Asteraceae</td>
<td>Stem</td>
<td>Drying stem is crushed mixed with Capsicum annuum and salt and drunk</td>
<td>Oral</td>
<td>Traditional Healer</td>
</tr>
<tr>
<td>18</td>
<td>Embelliaschimperi vatke</td>
<td>Myrsinaceae</td>
<td>Fruit</td>
<td>Crushed and drunk</td>
<td>Oral</td>
<td>Traditional Healer</td>
</tr>
<tr>
<td>19</td>
<td>Eucalyptus globulus</td>
<td>Myrtaceae</td>
<td>Leaf</td>
<td>Young leaves are chopped and boiled with water and breathe in the vapor</td>
<td>Nasal</td>
<td>Home Garden</td>
</tr>
<tr>
<td>20</td>
<td>Feoniculum vulgare</td>
<td>Apiaceae</td>
<td>Leaf</td>
<td>Fresh leaf soaked, mixed with milk and drunk</td>
<td>Oral</td>
<td>Home Garden</td>
</tr>
<tr>
<td>21</td>
<td>Ficus sur Forsk.</td>
<td>Moraceae</td>
<td>Fruit</td>
<td>Fruit juice is taken</td>
<td>Oral</td>
<td>Market</td>
</tr>
<tr>
<td>22</td>
<td>Hagenia abyssinica</td>
<td>Rosaceae</td>
<td>Leaf</td>
<td>Fresh leaves are powdered, mix with water and Dunk</td>
<td>Oral</td>
<td>Traditional Healer</td>
</tr>
</tbody>
</table>
Table 5. Contd.

<table>
<thead>
<tr>
<th>No.</th>
<th>Species</th>
<th>Family</th>
<th>Part</th>
<th>Disease</th>
<th>Preparation</th>
<th>Route</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>23</td>
<td>Hordeum vulgare</td>
<td>Poaceae</td>
<td>Gebis</td>
<td>Dandruff</td>
<td>Seeds are crushed and then mashed on infected part</td>
<td>Dermal</td>
<td>Market</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Wenagfit</td>
<td>Toothache</td>
<td>Dried leaves are powdered and added on teeth</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Neqarsa Root Roots are dried, powdered and mixed with tea</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Infected eye Leaf Fresh leaves chopped, pressed, and liquid is dropped into eye</td>
<td></td>
<td></td>
</tr>
<tr>
<td>24</td>
<td>Inula confertiflora A</td>
<td>Asteraceae</td>
<td>Wenagfit</td>
<td>Toothache</td>
<td>Wash the foot with fresh leaf</td>
<td>Dermal</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Kurba Leaf The leaves are crushed, squeezed and creamed on infected part</td>
<td></td>
<td>Home Garden</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Rheumatism Leaf The leaves are chopped and boil with water and immerse the infected part</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Dandruff Leaf The leaves are crushed, squeezed and creamed on infected part</td>
<td></td>
<td></td>
</tr>
<tr>
<td>25</td>
<td>Justicia schimperiana</td>
<td>Acanthaceae</td>
<td>Semiza</td>
<td>Foot fungi</td>
<td>The fresh leaf is heated and spread on the swollen part of the body</td>
<td>Dermal</td>
<td>Home garden</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Kurla Leaf The leaves are powder, mixed with water and sugar and drunk</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Rheumatism Leaf The leaves are chopped and boil with water and immerse the infected part</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Dandruff Leaf The leaves are crushed, squeezed and creamed on infected part</td>
<td></td>
<td></td>
</tr>
<tr>
<td>26</td>
<td>Kalancheo Shimperiana</td>
<td>Crassulaceae</td>
<td>Endahula</td>
<td>Body swelling</td>
<td>The fresh leaf is heated and spread on the swollen part of the body</td>
<td>Dermal</td>
<td>Traditional Healer</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Swelling Tonsil Root Fresh root is put on the nose</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Linear Placenta Seed are powdered and mixed water and salt and eaten with enjera</td>
<td>Oral</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Gastric Seed The seed are powdered, mixed with water and sugar and drunk</td>
<td></td>
<td>Market</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Diarrhea Seed The seeds are immersed in water and drunk</td>
<td></td>
<td></td>
</tr>
<tr>
<td>27</td>
<td>Linum usitatissimum L.</td>
<td>Lineaceae</td>
<td>Telba</td>
<td>Retained placenta</td>
<td>Seed are powdered and mixed water and salt and eaten with enjera</td>
<td>Oral</td>
<td>Market</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Gastric Seed The seed are powdered, mixed with water and sugar and drunk</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Diarrhea Seed The seeds are immersed in water and drunk</td>
<td></td>
<td></td>
</tr>
<tr>
<td>28</td>
<td>Lippia adoensis</td>
<td>Lamiaceae</td>
<td>Kesiye</td>
<td>Migh (fibri Illness)</td>
<td>The leaf and immature stem of this plant is ground, pounded and mixed with small amount of coffee and drunk</td>
<td>Oral</td>
<td>Home Garden</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Headache</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>29</td>
<td>Lepidium sativum L.</td>
<td>Brassicaceae</td>
<td>Feto</td>
<td>Toothache</td>
<td>Smoking the seeds on fire</td>
<td>Oral</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Diarrhea Seed Powdered seeds and garlic are mixed with honey and eaten</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Tonsillities Seed Powdered seeds and garlic are mixed with honey</td>
<td></td>
<td></td>
</tr>
<tr>
<td>30</td>
<td>Lepidium sativum L.</td>
<td>Brassicaceae</td>
<td>Feto</td>
<td>Cough</td>
<td>Powdered seeds and garlic are mixed with honey</td>
<td>Oral</td>
<td>Traditional Healer</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Wound Seed Powdered seeds mixed with water and apply in the wounded area</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Diarrhea with blood Seed Powder of feto mixed with milk, filter and drunk</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Hemorrhoids Stem Stem is heated and put on the infected part</td>
<td></td>
<td></td>
</tr>
<tr>
<td>31</td>
<td>Lupinus albus L.</td>
<td>Fabaceae</td>
<td>Gibto</td>
<td>Hypertension</td>
<td>Soaking seeds with water for 5 days, and eating softened seeds and/or preparing Areki( alcohol ) by using seeds and drunk.</td>
<td>Oral</td>
<td>Market</td>
</tr>
<tr>
<td>32</td>
<td>Moringa tenopetala</td>
<td>Moringaceae</td>
<td>Shiferaw</td>
<td>Blood pressure</td>
<td>Leaves are crushed and powdered, Mixed with tea and drunk</td>
<td>Oral</td>
<td>Market</td>
</tr>
<tr>
<td>Table 5. Contd.</td>
<td></td>
<td></td>
<td></td>
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<td>33 Nigella sativa L. Apioaceae Tikur azmuid</td>
<td>Headache, Asthma</td>
<td>Seed</td>
<td>Seeds are put in boiling water and steam is inhaled</td>
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<td></td>
<td>Sudden disease</td>
<td>Seed</td>
<td>Crushed seed drunk</td>
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<tr>
<td>34 Ocimum basilicum Lamioaceae Besoblia/Zigaqibey</td>
<td>Sudden sickness</td>
<td>Leaf</td>
<td>Chewing the fresh leaf</td>
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<tr>
<td>35 Ocimum lamifolium Lamioaceae Demakessi</td>
<td>Febrile illness (mich)</td>
<td>Leaf</td>
<td>Fresh leaf and stem are boiled in water and inhaled by vapor</td>
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<td></td>
<td>Coughs and colds</td>
<td>Leaf</td>
<td>Fresh leaves are squeezed and sniff the liquid</td>
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<td></td>
<td>Eye Disease</td>
<td>Leaf</td>
<td>Apply droplets leaf juice</td>
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<td></td>
<td>Headache</td>
<td>Leaf</td>
<td>Fresh leaf and stem are boiled in water and inhaled by vapor</td>
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<td></td>
<td>Mouth burns</td>
<td>Leaf</td>
<td>Fresh leaves are squeezed and applied on infected part</td>
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<td>36 Olea europaea Oleaceae Woyira</td>
<td>Wound</td>
<td>Leaf</td>
<td>Oil extracted from leaf is dropped on the wound area</td>
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<td>37 Otostegia integrifolia Lamioaceae Tenguit</td>
<td>Stomachache</td>
<td>Leaf</td>
<td>Fresh leaf juice is extracted and used</td>
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<td></td>
<td>Shotelay</td>
<td>Root</td>
<td>The root is tied on neck</td>
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<td></td>
<td>Rabis</td>
<td>Leaf</td>
<td>The leaf is crushed mixed with milk and drunk</td>
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<tr>
<td>38 Perisa americana Lauraceae Avocado</td>
<td>Anemia, blood pressure</td>
<td>Fruit</td>
<td>Extracted juice is taken</td>
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<td>39 Plantago lanceolata Plantaginaceae Gorteb</td>
<td>Fresh Wound</td>
<td>Leaf</td>
<td>Fresh leaves are crushed and spread over on wound</td>
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<td>40 Plumbago zeylanica L. Plumbaginaceae Amera</td>
<td>Snake poison</td>
<td>Leaf</td>
<td>Smoking leaves on fire</td>
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<td></td>
<td>Wound</td>
<td>Root</td>
<td>Powdered dried roots and applied of infected wound</td>
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<td></td>
<td>Hemorrhoid</td>
<td>Root</td>
<td>Anal</td>
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<td></td>
<td>Heart disease</td>
<td>Leaf</td>
<td>Oral</td>
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<td>41 Phytoleca dodecandra L. Phytolacaceae</td>
<td>Gonorrhea</td>
<td>Leaf</td>
<td>Crushed leaves are filtered and drunk</td>
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<td></td>
<td>Rabis</td>
<td>Root/Leaf</td>
<td>Fresh root or leaf extract is mixed with milk and taken</td>
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<td></td>
<td>Kuriba</td>
<td>Root</td>
<td>Fresh root is crushed, squeezed and mixed with little water and drunk</td>
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<td>42 Ricinus communis Euphorbiaceae Chakma/Gulo</td>
<td>Infected wound</td>
<td>Fruit, leaf</td>
<td>Leaves are smashed applied on wound</td>
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<td>43 Rhamnus prinoides L Rhamnaceae Gysho</td>
<td>Skin disease/infection</td>
<td>Leaf</td>
<td>Washing the infected body part by fresh leaves</td>
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<td></td>
<td>Liver</td>
<td>Root</td>
<td>Fresh root is powdered and mixed with water and drunk</td>
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<td></td>
<td>Tonsilitis</td>
<td>leaf</td>
<td>Its leaves with the leaves of Ruta chalpens is chopped together, mixed with drop of Citrus limon boiled and drunk</td>
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<td>44 Rosa abyssica Rosaceae Kega Hypertension ion</td>
<td>Its fruit is are ground, powdered, mixed with water and drunk</td>
<td>Fruit</td>
<td>Oral</td>
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<td>45 Ruta chalpens L Rutaceae Tena Adam Evil Eye</td>
<td>The fresh leaves mixed with garlic is crushed and Tie by neck and smelled by nose</td>
<td>Leaf</td>
<td>Nasal</td>
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nearby community are able to create strong bond with the community and trust each other in curing the disease and other social settings.

**Conclusion**

The study indicated that a total of 55 medicinal plant species belonging to 35 families were used to treat various human diseases. Majority of medicinal plants (80%) species were obtained by
cultivation in home garden. 48% of respondents have medicinal plants in their home garden. Leaf (13.3%) and root parts (13.3%) are the most widely used plant part for drug preparation.

Liquid (46.9%) is a widely used remedy preparation form. Oral method of administration accounts for 36.7% followed by dermal application (30%) and dermal and nasal in combination (20%). In general, Debare Markos Town is rich in sources. The practice of the use of traditional medicine is common in the study area. Most of the available medicinal plants are found under threats in the study area, which is one of the main reasons for the degradation and destruction of habitats is a major cause of the loss of medicinal plant. Therefore, documentation medicinal plants provide important data. The practice of using medicinal plants by the local people has enormous and paramount significant in economic and social sense. While practicing cultivating medicinal plants, they feel confident that they will cure different diseases within their compound. Especially, there are different infectious diseases and accidental illness that cause psychological and physical damage to the people and its being treated by those medical plants. Moreover, those people who used medicinal plants for the sake of their health can safe and reduce frequency of unnecessary waste from transportation and other miscellaneous cost to modern health institutions. Thus, it enhances strong economic capacity of the people through creating healthy, physically and mentally capable people.

ACKNOWLEDGEMENTS

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CONFLICT OF INTERESTS

The authors have not declared any conflict of interests.

REFERENCES


