INTRODUCTION

Populations of all the continents use plants for their various needs in terms of shelter, clothing, food, flavours and fragrances as not the least, medicines [1]. The last decade has witnessed a growing number of populations who rely on traditional remedies derived from medicinal plant and tremendous progress in medicinal plant research. This worldwide trend towards the use of natural plant remedies has created an enormous need for information about the properties and uses of the medicinal plant for the treatment of minor ailments to more complex diseases.

Malvaceae botanical family consists of dicotyledonous plants with at least 2300 species in 200 genera [2]. Wissadula amplissima a member of this family is present throughout Africa from Cape Verde to Senegal and Ethiopia to South Africa [3]. In the central region of Burkina Faso, species belonging to this family are well known and used by traditional practitioners to treat several diseases. Ethnopharmacological studies report the use of this species in folk medicine to treat vomiting, severe diarrheas, mucosa, skin and bronchus inflammation; inflammatory diseases, fever and allergies. The roots of the plant are used to treat anemia, hemorrhages and as a fortifying [4].

Infammation is a complex defensive system of the body against any aggression. It involves in pathogenesis of diseases including infections, cancer, diabetes, atherosclerosis, arthritis. Several mediators such as cytokines (TNF α, IL6, IL 1β), free radical (reactive oxygen species (ROS), prostaglandins (PGE2) and leukotriene) are released during inflammation response. Lipooxygenase (LOX) involved leukotriene synthesis play an important role in inflammation maintenance [5].

Previous biological investigations have demonstrated that methanol extract of Wissadula amplissima leaves possess antioxidant properties (2, 2-diphenyl-1-picrylhydrazyl method) and anti-inflammatory capacity using carrageenan-induced paw edema in chicks [6]. In the best of our knowledge, there is no scientific information concerning the in vitro anti-inflammatory activities using the enzymatic method, in vivo anti-inflammatory activities using carrageenan-induced paw edema in mice, the toxicity and analgesic activities of this species.

The aim of this present study was to investigate anti-inflammatory (in vivo and in vitro), analgesic and antioxidant effects of the hydroacetonic extract (HAE) of Wissadula amplissima whole plant and to determine polyphenol and flavonoids contents.

MATERIALS AND METHODS

Plant materials

The whole plant of Wissadula amplissima var. Rostrata (Shum. and Thonn.) R. E. Fries (Malvaceae) was harvested in January 2013 at Gampela (25 km, East of Ouagadougou, Burkina Faso). Botanical identity was assessed by Professor Jeanne Millogo-Rasolodimby from laboratoire de biologie et ecologie vegetale (University of Ouagadougou, Burkina Faso) where a voucher specimen (C I: 16884) was deposited. Plant material was dried at room temperature, pulverized and stored in an airtight bag until use.

Animals and housing

Naval medical research institute (NMRI) mice (7 to 8 w old, 25 to 35g body weight), provided by the animal housing facility of the Naval medical research institute (NMRI) mice (7 to 8 w old, 25 to 35g body weight), provided by the animal housing facility of the
University of Ouagadougou, were used. Mice were kept in an environmentally controlled breeding room (20-25°C, 75% humidity, 12 h photoperiod), fed with standard laboratory food and water ad libitum. Mice were fasted 17 h before experiments. Investigation at animals was conducted in accordance with the official journal of European committee in 1991 and approved by the institutional committee regarding the care and use of animals for the experimental procedure in 2010; (CEES09 [7, 8]).

**Chemicals**

Chemicals were from analytical grade. Acetic acid, acetylsalicylic acid, allopurinol, carrageenan, dimethylsulfoxide, 2,2-diphenyl-1-picyrylhydrazyl, hydrocortisone, lecithin, 15-lipoxygenase (EC 1.1.3.22), paracetamol, quercetin, sodium chloride, xanthine and xanthine oxidase (EC 1.1.3.22) were purchased from Sigma-Aldrich (St. Louis, MO, USA). Ascorbic acid, desoxypirrole, ferric trichloride, hydrochloric acid, potassium hexacyanoferrate, thioarbituric acid and trichloroacetic acid were supplied by Labosi (Paris, France). Boric acid, ethylenediamine tetraacetic acid, gallic acid, linoleic acid, sodium carbonate, sodium phosphate dibasic, sodium phosphate monobasic, sodium tetraborate, trichloroacetic acid and all solvent used were sourced from Probalo (Paris, France).

**Extract preparation**

Powdered plant material (50 g) was defatted with petroleum ether (500 ml) in a soxhlet extractor and then soaked (48 h, 25 °C, continuous stirring) in acetone/water (80/20, 500 ml). Extract was filtrated, concentrated in a vacuum evaporator (Buchi Rotavapor R-200) and lyophilized (Telstar Cryodos 50) to get 44.7 g of hydro acetone extract (HAE). HAE was stored at 4 °C until use.

**Acute toxicity test**

Acute toxicity was carried out to determine lethal dose (LD₅₀). NMRI mice orally received 500, 1000, 2000, 3000, 5000 mg/kg and intraperitoneally 1000 and 1500 mg/Kg of body weight (bw). After the administration of extract, the animals were observed for 2 to 72 h. Then morbidity or mortality and changes in behavior have been noted [9].

**Anti-edematous activity**

Anti-edematous activity was evaluated according to Winder et al. [10]. Seven groups of six mice were randomly used. HAE of Wissadula amplissima was administrated per os at 2 different doses (200 and 300 mg/kg bw) and intraperitoneally at 150 mg/kg bw to sample groups. Control groups received only vehicle (Water per os, 0.9 % NaCl ip) while reference groups received reference compounds (acetylsalicylic acid at 150 mg/kg bw, hydrocortisone at 10 mg/kg bw ip). One hour later, 50 µl of carrageenan (1% in 0.9% NaCl) was injected under the aponeurosis of plantar of the hind paw at each mouse. Paw edema volume was measured at time intervals of 1 h, 3 h and 5 h after carrageenan injection using plethysmometer (model Ugo Basili, n =7141, Italy). The anti-edematous activity was evaluated in the percentage of reduction of the edema in treated mice compared to negative controls using the following formula:

\[
\% \text{Inhibition} = \frac{A-B}{A} \times 100
\]

A represents the average difference paw edema volume in the negative control group and B represents the average difference of paw edema volume in treated groups of mice.

**Analgesic activity**

The analgesic activity was evaluated according to the acetic acid-induced writhing test as described by Sawadogo et al. [11] with minor modifications. Six groups of 6 randomly selected mice were used. HAE of Wissadula amplissima was administrated per os at 2 different doses (200 and 300 mg/kg bw) and intraperitoneally at 150 mg/kg by to sample groups. Control groups received only vehicle (Water per os, 0.9 % NaCl ip) while reference group received paracetamol (100 mg/kg bw per os). One hour after drug administration, mice received 0.6% of acetic acid injection (10 ml/kg bw). A number of writhing was recorded within 5 to 20 min after acetic acid injection. The analgesic effect was evaluated according to the following formula:

\[
\text{% Inhibition} = \frac{\text{Number of Writhes}_{\text{Control}} - \text{Number of Writhes}_{\text{Sample}}}{\text{Number of Writhes}_{\text{Control}}} \times 100
\]

**Lipoxygenase (LOX) inhibitory activity**

The inhibition of lipoxygenase was assayed according to the spectrophotometric method described by Lyckander and Malterud [12] with slight modifications. Briefly, 100 µl of enzyme solution (200 U/ml) prepared in boric acid buffer (0.2 M, pH 9.0) was mixed with 25 µl of HAE of Wissadula amplissima (1 mg/ml in DMSO) and then incubated at room temperature for 3 min. Reaction was then initiated by the addition of 125 µl of the substrate (250 µM of linoleic acid) and the velocity was recorded for 3 min at 234 nm with a microplate reader (Epoch, BioTek instruments, USA). DMSO was used as a control while quercetin and ibuprofen were used as reference compounds. Percentage of lipoxygenase inhibition was calculated according to the equation:

\[
\text{% Inhibition of lipoxygenase} = \frac{\text{V}_{\text{Control}} - \text{V}_{\text{Sample}}}{\text{V}_{\text{Control}}} \times 100
\]

**DPPH radical scavenging assay**

DPPH radical scavenging activity was evaluated as described by Kadam et al. [14]. Briefly, 200 µl of freshly prepared DPPH solution (0.02 mg/ml in methanol) was mixed with 100 µl of HAE of Wissadula amplissima from 100 to 25 µg/ml in methanol. After shaking the mixture was incubated for 15 min in darkness at room temperature and absorbance measured at 517 nm against a blank (methanol) with a microplate reader (Epoch, BioTek instruments, USA). Inhibition of DPPH radical was calculated as following:

\[
\text{% Inhibition of DPPH radical} = \frac{\text{Abs}_{\text{Blank}} - \text{Abs}_{\text{Sample}}}{\text{Abs}_{\text{Blank}}} \times 100
\]

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

FRAP assay was performed according to Lamién-Meda et al. [15]. HAE of Wissadula amplissima (100 µl 1 mg/ml in methanol) was mixed with 250 µl of phosphate buffer (0.2 M, pH 6.6) and 250 µl of potassium hexacyanoferrate solution (1% in water). After 30 min of incubation (50 °C), 250 µl of trichloroacetic acid (10% in water) was added and the mixture was centrifuged (2000 g for 10 min). The supernatant (125 µl) was mixed with water (125 µl) and 25 µl of fresh FeCl₂ solution (0.1 % in water) and then absorbance was read at 700 nm with a microplate reader (Epoch, BioTek instruments, USA).
USA). Ascorbic acid was used to plot a calibration curve \((R^2 = 0.99)\). Reducing power was expressed as mg ascorbic acid equivalent per gram of HAE (mg AAE/g). Gallic acid and quercetin were used as positive controls.

**Lipid peroxidation inhibition assay**

Inhibition of lipid peroxidation was determined following the method described by Kulkarni et al. [16]. Briefly, 100 µl of HAE of *Wissadula amplissima* (from 100 to 25 µg/ml in methanol) were added to 100 µl of 1% a-Tocopherol (10 mmol/ml) and 5 min after 1-hour incubation at 37 °C, HCl (1 ml, 0.25N) and thiobarbituric acid (1 ml of 1% aqueous solution) and incubated at 37 °C for 1 hour with HAE (100 µl; 1 mg/ml). The total thiobarbituric acid reactive substances was determined according to the method described by Kulkarni et al. [16].

Desoxyribose degradation assay

The ability of HAE of *Wissadula amplissima* to prevent the degradation of the desoxyribose was determined using the method described by Houghton et al. [17]. Briefly, freshly prepared 2-desoxyribose (100 µl; 28 mmol in phosphate buffer 50 mmol, pH 7.4), EDTA (500 µl; 1.04 mmol), FeCl₃ (100 µl; 1.0 mmol) and ascorbic acid (100 µl; 20 mmol) for 1 hour incubation at 37 °C, HCl (1 ml, 0.25N) supplemented with 15% TCA and 0.375% TBA was added to the mixture, incubated for 15 min at 100 °C and centrifuged (3000 rpm for 10 min). The absorbance of the supernatant was read at 532 nm against a blank (methanol) with a microplate reader (Epoch, BioTek instruments, USA). Inhibition of lipid peroxidation was calculated as following:

\[
\% \text{Inhibition of lipid peroxidation} = \frac{Abs_{\text{blank}} - Abs_{\text{sample}}}{Abs_{\text{blank}}} \times 100
\]

Abs blank and Abs sample are the absorbance of the blank and sample reactions. IC₅₀ (Concentration inhibiting 50% of lipid peroxidation) was graphically determined. Quercetin was used as a positive control.

**Desoxyribose degradation assay**

The total polyphenol content was determined according to Singleton et al. [18]. HAE of *W. amplissima* (25 µl, 100 µg/ml in Methanol) was mixed with aluminum trichloride (75 µl, 2% in methanol). Absorbance was subsequently read at 415 nm after 10 min incubation against a blank with a microplate reader (Epoch, BioTek instruments, USA). A standard calibration curve \((y = 0.0029x + 0.0036; R^2 = 0.99)\) was plotted using Quercetin (0-100 mg/l). Flavonoid content was expressed as mg of Quercetin equivalent to 100 mg of extract (mg QE/100 mg).

### Statistical analysis

Experiments were carried out in triplicate and results expressed as mean±SEM. ANOVA analysis (p<0.05) and linear regression were performed with GraphPad Prism 5 and XLSTAT 7.1 software.

**RESULTS**

**Acute toxicity**

Any mortality was observed at the highest doses of *W. amplissima* HAE (5000 mg/Kg bw per os, 1500 mg/Kg bw intraperitoneally) on mice model. However behavioral signs (prostration, loss of exploration instinct, sleepiness and feed refusal) were observed within two hours following the extract administration per os at 5000 mg/kg bw. Although behavioral signs disappear after 2 d mice regain their initial vitality. These results point out that HAE did not exhibit any acute toxicity effect on NMRI mice at the doses tested.

**Anti-inflammatory activity in vivo**

The anti-inflammatory activity of *W. amplissima* HAE was assessed in vivo through its anti-edematous and analgesic effects as summarized in table 1 and table 2.

* Either in oral or intraperitoneal (i. p.) administration, edema induced by carrageenan injection was significantly (p<0.05) reduced in a time-dependent manner within 5 h after inflammation, that is not the case of acetylsalicylic acid administrated per os. In our experimental conditions, HAE administrated per os at 200 and 300 mg/kg bw were less active than acetylsalicylic acid (150 mg/kg bw per os) within an hour but were more efficient at the fifth hour. HAE (150 mg/kg, i. p.) exhibited the same effect than hydrocortisone (15 mg/kg) within 3 h, the latter being more effective at the fifth hour. Regarding the analgesic activity, reduction of contortions dose-dependent when HAE is administrated per os. Either in oral (200 and 300 mg/kg bw) or intraperitoneal (150 mg/kg bw) administration HAE was more effective than paracetamol (100 mg/kg, bw, per os).

**Inhibition of lipoygenase and xanthine oxidase**

Table 3 show that HAE possess significant inhibitory activities on both enzymes respectively with 72.8% inhibition for xanthine oxidase (XO) and 68.99% inhibition for lipoygenase (LOX) when tested at 100 µg/ml (final concentration). Regarding LOX inhibition, HAE is more potent than quercetin (52.97%) but less active than ibuprofen (78.61%). Concerning XO inhibition, HEA is slightly weaker than quercetin (93.63%) and allopurinol (95.49%).

### Table 1: Effect of HAE of *W. amplissima* on edema induced in mice by the injection of carrageenan

<table>
<thead>
<tr>
<th>Samples administration (mg/kg)</th>
<th>Volume of edema (ml)</th>
<th>Inhibition percentage (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1 h</td>
<td>3 h</td>
</tr>
<tr>
<td></td>
<td>1 h</td>
<td>3 h</td>
</tr>
<tr>
<td><strong>per os</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control (NaCl 0.9%)</td>
<td>0.21±0.04</td>
<td>0.33±0.01</td>
</tr>
<tr>
<td>HAE (200)</td>
<td>0.16±0.01</td>
<td>0.19±0.01*</td>
</tr>
<tr>
<td>HAE (300)</td>
<td>0.13±0.03*</td>
<td>0.23±0.03*</td>
</tr>
<tr>
<td>acetylsalicylic acid (150)</td>
<td>0.10±0.01*</td>
<td>0.16±0.01*</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>i. p.</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control (NaCl 0.9%)</td>
<td>0.22±0.05</td>
<td>0.36±0.03</td>
</tr>
<tr>
<td>HAE (150)</td>
<td>0.15±0.05*</td>
<td>0.21±0.01*</td>
</tr>
</tbody>
</table>

Data are expressed as mean values±SEM (n = 6). HAE: Hydro acetone extract of *W. amplissima*. *Values within the same sub-column harbouring asterisk mark in superscript are significantly different from the control group (p<0.05).
Table 2: Effect of HAE of *W. amplissima* against writhing induced by acetic acid

<table>
<thead>
<tr>
<th>Samples administration (mg/kg)</th>
<th>Number of Writings</th>
<th>Inhibition percentage (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>i.p.</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>66.27±6.50</td>
<td>---</td>
</tr>
<tr>
<td>HAE (200)</td>
<td>28.66±1.47*</td>
<td>56.74±2.41</td>
</tr>
<tr>
<td>HAE (300)</td>
<td>24.66±4.84*</td>
<td>62.78±6.67</td>
</tr>
<tr>
<td>Paracetamol (100)</td>
<td>35.16±2.13*</td>
<td>46.94±2.78</td>
</tr>
<tr>
<td><strong>i. p.</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>74.40±3.79</td>
<td>---</td>
</tr>
<tr>
<td>HAE (150)</td>
<td>47.93±3.67*</td>
<td>47.93±3.67</td>
</tr>
</tbody>
</table>

Data are expressed as mean ± SEM (n = 6). HAE: Hydro acetone extract of *W. amplissima*. Values within the same sub-column harboring asterisk mark in superscript are significantly different from the control group (p < 0.05).

Table 3: Inhibition of xanthine oxidase and lipoxygenase by HAE from *W. amplissima*

<table>
<thead>
<tr>
<th>Samples (µg/ml)</th>
<th>Xanthine oxidase inhibition (%)</th>
<th>Lipoxygenase inhibition (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>HAE (100)</td>
<td>72.80±1.44*</td>
<td>68.99±13.9*</td>
</tr>
<tr>
<td>Allopurinol (100)</td>
<td>95.49±0.47*</td>
<td>nd</td>
</tr>
<tr>
<td>Quercetin (100)</td>
<td>93.63±0.36*</td>
<td>52.97±0.71*</td>
</tr>
<tr>
<td>Ibuprofen (100)</td>
<td>nd</td>
<td>78.61±1.12*</td>
</tr>
</tbody>
</table>

Data are expressed as mean ± SEM (n = 3). HAE: Hydro acetone extract of *W. amplissima*. Within the same column, values with different letter in superscript are significantly different (p < 0.05).

Antioxidant activity, polyphenol and flavonoid content

The antioxidant potential of the HAE of *W. amplissima* to scavenge DPPH radical, to prevent ferric reduction, lipid peroxidation and desoxyribose degradation has been compared with those of Quercetin. As shown (table 4), HAE of *W. amplissima* demonstrates an interesting antioxidant potential. Indeed, in the lipid peroxidation model, HAE of *W. amplissima* exhibited the same antioxidant activity than quercetin, our antioxidant reference. Although HAE of *W. amplissima* was less active than quercetin in the other antioxidant model tested, its ferric reducing power was only 1.5 lower than that of quercetin and IC₅₀ values recorded in DPPH and desoxyribose assays were lower than 100 µg/ml. Total polyphenol and flavonoid content of HAE from *W. amplissima* were respectively 64.33±1.34 GAE/100 mg of extract and 35.17±0.77 QE/100 mg of extract.

Table 4: Antioxidant activity, polyphenol and flavonoid content of HAE from *W. amplissima*

<table>
<thead>
<tr>
<th>Samples (µg/ml)</th>
<th>Xanthine oxidase inhibition (%)</th>
<th>DPPH assay (IC₅₀, µg/ml)</th>
<th>Desoxyribose assay (IC₅₀, µg/ml)</th>
<th>Lipid peroxidation assay (IC₅₀, µg/ml)</th>
<th>FRAP assay (µmol AAE/g)</th>
<th>Polyphenol content (mg GAE/100 mg)</th>
<th>Flavonoid content (mg GAE/100 mg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>HAE</td>
<td>57.33±2.3*</td>
<td>32±2.51*</td>
<td>11.9±0.1*</td>
<td>120.37±2.5*</td>
<td>64.33±1.34</td>
<td>35.17±0.77</td>
<td></td>
</tr>
<tr>
<td>Quercetin</td>
<td>1.35±0.13*</td>
<td>10.06±0.11*</td>
<td>9.13±0.11*</td>
<td>187.77±2.27*</td>
<td>35.17±0.77</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Ibuprofen</td>
<td>1.35±0.13*</td>
<td>10.06±0.11*</td>
<td>9.13±0.11*</td>
<td>187.77±2.27*</td>
<td>35.17±0.77</td>
<td>-</td>
<td></td>
</tr>
</tbody>
</table>

Data are expressed as mean ± SEM (n = 3). HAE: Hydro acetone extract of *W. amplissima*. Within the same line, values harboring different letter in superscript are significantly different (p < 0.05).

DISCUSSION

The present study demonstrated that hydro acetic extract (HAE) of *W. amplissima* possess potential pharmacological properties including antioxidant, anti-edematous, analgesic and inhibiting enzymes (xanthine oxidase and lipoxygenase) effects. HAE of whole *W. amplissima* was considered as slightly toxic conferring to toxicity scale established by Hodge and Sterner [9], because the LD₅₀ was above 5000 µg/kg of body weight.

Edema induced by the carraganan injection is an animal model assay commonly used to evaluate the anti-inflammatory activity of substances. Carraganan injection provokes the release of several chemical mediators which are the basis of the inflammatory process. The inflammatory response is biphasic [20, 21]: the first phase lasted one hour that mediated by histamine, serotonin and bradikynin; the second phase (above one hour) is due to the biosynthesis of prostaglandins [22]. HAE of *W. amplissima* whole plant inhibited the edema in a dose-dependent manner and in all these phases. Thus, the extract may have an antagonistic action against histamine, bradikynin, serotonin and the prostaglandins biosynthesis. The highest value of inhibition was obtained at the fifth hour. This suggests that the inhibitor action of the HAE of *W. amplissima* could be act more on the cyclooxygenases which are implicated in the synthesis of prostaglandins.

The extract was effective as well as it was orally administrated or by intraperitoneal injection. The molecular structure plays a key role in the choice of the way used to administrate a drug. In this study results shown that whatever the administration mode was (orally or intraperitoneally) the HAE of *W. amplissima* was effective. This suggests that bioactive compounds, responsible for the anti-inflammatory properties found in this herbal mixture have been easily absorbed through the intraperitoneal way or via the gastrointestinal mucosa.

Writhing induced by acetic acid injection is a method used to study the peripheral analgesic effect of a substance [23]. Abdominal contortions induced by acetic acid injection were used to evaluate the analgesic effect of HAE of *W. amplissima*. Test showed that the extract had a non-opioid analgesic potential either oral or intraperitoneal administration. The pain caused by the injection of acetic acid is due to the release of serotonin, bradikynin, substance P and prostaglandins (PGE₅₀ and PGF₅₀). These chemical mediators stimulate peripheral nociceptive neurons and induce the vascul permeability [24]. HAE of *W. amplissima* inhibited abdominal constriction in a dose-dependent manner. Analgesic effect of HAE of *W. amplissima* could be related to the inhibition of these chemical mediators.

Lipoxygenases (LOXs) play an important role in the biosynthesis of leukotrienes which are pro-inflammatory mediators involved in the physiopathology of inflammatory diseases. Leukotriene LTB₄ is a powerful pro-inflammatory chemotactic agent for a variety of leukocytes; and leukotrienes (CA, D₄, E₄) are responsible of vascular permeability and smooth muscle contraction [25]. HAE of *W. amplissima* inhibited lipooxygenase; it suggests that extract prevents leukotrienes actions inhibiting LOX.
The enzyme xanthine oxidase (XO) catalyzes the oxidation of hypoxanthine and xantine to uric acid in the purine catabolic pathway [26]. Uric acid plays a crucial role in pathogenesis hyperuricemia, cardiovascular diseases [27]. HAE of W. amplissima whole plant inhibited XO. HAE of W. amplissima whole plant was evaluated for its in vitro anti-inflammatory activity by inhibition of LOX and XO enzymes. The inhibition of these enzymes is a potential therapeutic to treat asthma, hyperuricemia and gout. 

During inflammatory process, excessive production of free radical such as reactive oxygen species (ROS) are released causing cell lysis, pro-inflammatory cytokines (TNFα, IL1β, IL6) production and LOX and cyclooxygenases expression [28]. The inhibitor effect of HAE of W. amplissima whole plant against ROS, was evaluated using DPPH, desoxyribose degradation assay, FRAP and lipid peroxidation methods that were widely used to determine the antioxidant capacity of substances. The extract exhibited antioxidant activity with four methods, however, its effect was smaller than quercetin. In this study different methods were used because it takes more than one antioxidant method to assess the mode of action of antioxidants [29]. The antioxidant capacity of W. amplissima HAE contributes to reinforce its anti-inflammatory effect.

The results of this study reveal that HAE of W. amplissima contains polyphenol and flavonoid compounds which could be responsible of evidenced pharmacological activities.

CONCLUSION

The present study showed that the hydroacetonic extract of Wissadula amplissima possesses antioxidant, analgesic and anti-inflammatory (in vitro and in vivo) effects. Data obtained represent a scientific basis for the traditional use of Wissadula amplissima to manage inflammatory diseases. Further bio-guided investigations would help in the isolation of compounds and in the clear elucidation of their mechanism of action.

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AUTHORS CONTRIBUTIONS

All the author have contributed equally

CONFLICT OF INTERESTS

Authors have no conflict of interest to declare

REFERENCES