

Analgesic, Non-ulcerogenic and Antioxidant Activities of the Aqueous Stem Bark Extract of *Morinda lucida* (Rubiaceae) in Mice

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Authors' contributions

This work was carried out in collaboration between all authors. Authors MC and BBP designed the study and wrote the protocol. Authors MC, MMG and TFS managed the analgesic tests. Authors MC, AAP and MMG managed the gastric ulcer tests. Authors BBP and TFL managed the antioxidant tests. Authors BBP and TFL did the phytochemical analysis. Authors MC and MMG did the literature search and statistical analysis. Authors MC and AAP wrote the first draft. Author TPV supervised the study. All authors read and approved the final manuscript.

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ABSTRACT

Aims: The objective of this study was to evaluate the analgesic; non ulcerogenic and antioxidant effects of the aqueous stem bark extract of *Morinda lucida* in mice.

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Materials and Methods: The phytochemical sifting was realized in order to find the chemical composition of the aqueous stem bark extract of *Morinda lucida* (ASBEML). The antioxidant activity was evaluated by the measurement of the capacity of ASBEML to trap radical DPPH, to reduce Fe^{3+} , and measure its total polyphenols. The effects of ASBEML were tested on models of acute pain induced by acetic acid and formalin in mouse fasted since 12 hours. The ulcerogenic effect of this plant was evaluated by administration of different doses of the aqueous extract to mouse fasted since 48 hours.

Results: The phytochemical tests revealed the presence of phenolics, flavonoids, catechic tannins, alkaloids, saponins and triterpenes. The DPPH, FRAP and Folin-Ciocalteu tests showed that the aqueous stem bark extract of *Morinda lucida* has a significant antioxidant activity. The oral administration of ASBEML significantly and dose dependently reduced the number of abdominal constrictions induced by injection of acetic acid. A maximum inhibition of 80.20% was obtained at the dose of 400 mg/kg. Also, this extract (400 mg/kg) significantly and dose dependently inhibited both phases of pain induced by formalin injection; the first phase was reduced up to 53.7% while the second phase was reduced up to 75.88%. The oral administration of ASBEML doesn't cause gastric ulcer. On the contrary, it significantly and dose dependently stimulated the mucus secretion.

Conclusion: In sum, the ASBEML possesses antioxidant, analgesic (centrally and peripherally) and non-ulcerogenic properties.

Keywords: *Morinda lucida*; Rubiaceae; antioxidant; pain; ulcer; mucus.

1. INTRODUCTION

According to the International Association for the Study of Pain (IASP), pain is an unpleasant sensory or emotional experience, linked to a real or potential tissue injury that causes protective motor and vegetative reactions, leading to individual modification behavior [1]. If acute pain is a warning signal that allows the individual to escape from the source of pain (stimulus), chronic pain is a severe disabling illness very often associated with other conditions such as osteoarthritis, rheumatism, cancer and many others. The consequences of chronic pain are very numerous, both organically and psychologically, with a behavioral modification that can go as far as anxiety-depression and an increased risk of suicide [2]. It is increasingly high in the world because of its multiple causes. According to Stucky et al. [3], over the third of the world's population suffers from chronic pain.

The monthly prevalence of pain of any kind is 72.40% in England [4]. According to Jain et al. [5], each person suffers from pain at least once in his life. Pain affects the entire world population and is therefore a major problem of public health. Considering the increasing prevalence, the difficulties in the management and the numerous consequences of the pain, this pathology requires special attention and an effective treatment to avoid possible complications.

However, most pharmaceuticals used in the treatment of pain have many side effects and some of them are relatively inaccessible to all segments of society. NSAIDs (such as indomethacin, ibuprofen) remain the most widely used therapeutic class in the world for the treatment of pain and inflammation, whether in the context of medical prescription or that of self-medication, because of their anti-inflammatory, antipyretic or analgesic properties and even inhibitory platelet aggregation [6]. However, these NSAIDs are responsible for a significant mortality generated mainly by their severe digestive adverse effects [7]. It is therefore essential to find new medicines available, less expensive, more effective and without side effects as far as possible.

Traditional medicine intervenes at this level. It is the first resort for primary health care, as about 80% of the population in developing countries is oriented towards traditional medicine, where 85% of treatments involve the use of plant extracts [8]. Plants are therefore a potential source of active ingredients that can lead to the development of new drugs. Moreover, 25 to 30% of the pharmaceutical products are of natural origin [9].

For example, *Morinda lucida* is a tree belonging to the Rubiaceae family [10]. The genus *Morinda* comprises about 80 species. In Africa, there are 5 species (*Morinda citrifolia*, *Morinda geminata*, *Morinda longiflora*, *Morinda morindoides* and

Morinda lucida). The relatively small floral and fruiting heads on long thin peduncles are distinctive characters of *Morinda lucida* [11]. *Morinda lucida* is a plant measuring on average 15 to 18 meters, some specimens up to 25 meters [12]. *Morinda lucida* is still called: sulfur tree. In Cameroon, *Morinda lucida* is called *ikeng* (Bassa), *akeng* (Beti), *keug* (Makia) and *didonedov* (Bamiléké). This tree is also known as *konkroma* (Twi) in Ghana, and *nfia* (Igbo in Nigeria) [13]. Previous phytochemical studies have revealed the presence of several classes of compounds in the aqueous extract of *Morinda lucida* bark such as: tannins, phenols, reducing substances, saponins, glycosides and flavonoids. Phytochemical screening revealed that fatty acids and alkaloids were absent [14]. The study of the acute and subacute toxicity of the aqueous extract of *Morinda lucida* stem bark has shown that this extract is weakly toxic [15].

Previous studies have shown that *Morinda lucida* has several pharmacological virtues. The leaf extract treats malaria, gastric ulcers and diabetes. The root bark extract is used in the treatment of diabetes and also has bactericidal activity. The methanol extract of trunk bark has anti-malarial, anti-cancer [16] and antioxidant effects [17]. While the work of Domekouo et al. showed that the aqueous extract of *Morinda lucida* bark has antidiabetic and antioxidant properties [14]. *Morinda lucida* is used in traditional medicine in the treatment of pain [18] and peptic ulcers [14]. The bark of *Morinda lucida* thus possesses analgesic effects without adverse effects on the gastric mucosa. This led us to evaluate the analgesic, non-ulcerogenic and antioxidant effects of ASBEML.

2. MATERIALS AND METHODS

2.1 Collection and Identification of Plant Material

The stem barks of *Morinda lucida* was harvested in August 2015 at Yaoundé (Center Cameroon region). Botanical identification was done in the National herbarium of Cameroon (Yaounde), by comparison with existing voucher specimens N° 2528 SRFK.

2.2 Preparation of Extract

In the laboratory, the barks were cut into pieces, dried under laboratory temperature and ground to powder. 1000 g was introduced in 5 liters of

distilled water and then boiled on a heating plate for 15 minutes. The resulting solution was filtered using a n° 3 Whatmann filter paper. The filtrate was lyophilized and the resulting brownish solid was used for the pharmacological tests. The resulting material weighed 37.50 g, giving a percentage yield of 3.75% with respect to the powder.

2.3 Animals

Adult male Swiss albino mice *Mus musculus* (25.00 ± 5.00 g) obtained from the Animal house, Laboratory of Animal Physiology, Department of Biological Sciences, Higher Teachers' Training College, University of Yaoundé I (Yaoundé-Cameroon) were used for this study. Prior authorization for the use of laboratory animals in this study was obtained from the Cameroon National Ethics Committee (Reg. No. FWAIRB00001954). The use, handling and care of animals were done in adherence to the European Convention (Strasbourg, 18.III.1986) for the protection of vertebrate animals used for experimental and other purposes (ETS-123), with particular attention to Part III, articles 7, 8 and 9.

2.4 Chemicals

Acetic acid, formalin, indomethacin and the aqueous extract were prepared prior to their use in the biological assays.

2.5 Phytochemical Tests

The aqueous stem bark extract of *Morinda lucida* was subjected to qualitative chemical test for the identification of different phytoconstituents like phenols, catechic tannins, galic tannins, flavonoids, alkaloids, saponins, triterpenes and sterols [19].

2.6 Measurement of *In vitro* Antioxidant Capacity of *Morinda lucida* Extract

2.6.1 Measurement of folin antioxidant capacity

Folin-Ciocalteu reagent (Sigma Chemical Co., St. Louis, MO) was diluted 10-fold and used for the measurement of the Folin antioxidant capacity of the extract [20], and absorbance was measured at 750 nm after 10 min. of reaction, using catechin as the standard.

2.6.2 Measurement of ferric reducing antioxidant power (FRAP)

The ferric reducing antioxidant ability of the extract was measured by spectrophotometry [21]. The FRAP reagent (2 mL) was mixed with 30 µL of hydrolysed extract and the absorbance was read at 593 nm after 12 minutes of incubation using a Spectronic GENESYS 5 incubator (Milton Roy Co.) equipped with a thermostat, autocell heating, and cooling water bath (Fischer Scientific). The temperature was maintained at 37°C and catechin was used as standard.

2.6.3 Measurement of DPPH scavenging activity

DPPH (1,1-diphenyl-2-picrylhydrazyl) radical scavenging activity of the extract was measured as earlier described [22]. An aliquot of the extract in methanol (10 mg/mL) was added to methanolic solutions of DPPH (1 mM, 0.25 mL). The mixture was shaken and left to stand for 30 minutes at room temperature. The absorbance of the solution was measured spectrophotometrically at 517 nm and the % DPPH scavenging activity was calculated against the control.

2.7 Investigating Analgesic Properties

2.7.1 Animal allotment and treatment

In each analgesic test, five groups of 6 animals were used. Group 1 served as negative control and received distilled water (0.5 ml/30 g bw). Group 2 was used as positive control and was treated with indomethacin (50 mg/kg per os). The 3 last groups received the aqueous extract of *Morinda lucida* at the doses 100; 200 and 400 mg/kg per os.

2.7.2 Acetic acid-induced writhing test

This test was conducted as previously described by Koster [23]. One hour after administration of the treatment, acetic acid solution (0.6v/v) was injected intraperitoneally (10ml/kg) to each animal. The number of writhing induced by the acetic acid, consisting of abdominal constrictions and hind limbs stretching were counted for 20 minutes after a latency period of 5 minutes. The percentage of inhibition was calculated as follows:

$$\text{Percentage of inhibition} = 1 - \frac{N_t}{N_c} \times 100$$

Where, N_c is the average number of stretching in the control group, N_t is the average number of stretching in the test group.

2.7.3 Formalin- induced pain

The method was conducted as previously described by Gaertner et al. [24]. In this procedure, 20 µL of 2.5% formalin was injected in the plantar arch of the right hind paw of the rats 1 hour after administration of drugs. These rats were individually placed in transparent cage for observation. The time spent licking the injected paw, was an indicator of the pain sensation following formalin administration, and was recorded in different phases: from 0-5 min post injection (first phase) and 15-30min post injection (second phase). These phases represented neurogenic and inflammatory pain response, respectively. The percentage of analgesic activity was calculated as follows:

$$\text{Percentage of inhibition} = 1 - \frac{T_t}{T_c} \times 100$$

With T_c is the mean time in control group for each phase and T_t is the mean time in the test group for each phase.

2.8 Investigating Non-ulcerogenic Properties

2.8.1 Ulcerogenic test

This test was assayed according to Grewal et al. [25]. Five hours after administration of drugs, mice were sacrificed under ether anaesthesia. The abdomen of each mouse was opened and stomach was located and removed after bindings at the levels of the cardia and the pylori. Each stomach was dilated by injection of 1 ml of formalin at 2% and was open along the greatest curve to examine macroscopically lesions.

2.8.2 Ulcer scores

The ulcers produced in the glandular region of each stomach were measured and scored as described by Martin et al. [26].

Description	Scores
No ulcers	0,0
Dilation of the vessels and small points of ulcers	1,0
Ulcers lower or equal to 4 mm length	2,5
Ulcers equal or higher to 5 mm length	5

2.8.3 Ulcer index (UI)

The ulcer index (UI) is the average score of ulcers of each treatment \pm the standard error on the mean (ESM) and calculated as follows:

$$IU = \sum_1^n \text{Scores} \pm \text{ESM}$$

2.8.4 Percentage of ulceration

The percentage of ulceration was calculated respectively to the ulcerated surface (US) (mm²) by the following formula:

$$\text{U.S.} = \left(\frac{\text{Total ulcerated surface}}{\text{Total surface of the stomach}} \right) \times 100$$

2.8.5 Determination of the mean area of the mice stomach

The mean area of the stomach of the mouse was determined as follows:

- In a number of 10 mice that received any treatment, the stomach was removed and opened.
- On the glandular part of each stomach was placed a millimetered paper soaked in oil (to make it more translucent) and the number of tiles inscribed was counted. The half-tiles were counted as whole but with only one side between the left and the right, and between the top and the bottom.
- The area of each stomach was calculated assuming that each tile was 1 mm² in area and the average of the ten surfaces was finally calculated; which allowed us to find an average surface area of 204.42 mm².

2.8.6 Measurement of mucus production

After estimating the degree of lesion formation, the gastric mucosa of each rat was immediately scraped gently using a glass slide and the mucus obtained was weighed using a precision electronic balance. The same experimenter performed this operation each time.

2.9 Statistical Analysis

Statistical analysis was done by one-way analysis of variance (ANOVA) followed by the

Tukey's post-test for multiple comparisons using Graphpad Prism 05 software and p values less than 0.05 were considered as significant. The results were expressed as mean \pm standard error of mean (SEM).

3. RESULTS

3.1 Phytochemical Characteristics of Extract

The result of preliminary phytochemical analysis shows that the aqueous stem bark extract of *M. lucida* possess catechic tannins, triterpenes, flavonoïds, alkaloïds, saponins and phenols. While sterols and galic tannins were absent (Table 1).

3.2 Antioxidant Activity

The ferric reducing antioxidant power (FRAP) of the aqueous stem bark extract of *M. lucida* was 994.53 \pm 6.11 mg/g ascorbic acid equivalent. Antioxidant capacity, measured as a function of the extract polyphenols content, was 299.89 \pm 1.62 mg/g ascorbic acid equivalents, while the percentage DPPH radical scavenging activity was 49.44.

3.3 Analgesic Properties

3.3.1 Acetic acid-induced writhing test

Aqueous stem bark extract of *Morinda lucida* strongly reduced writhing and stretching induced by the *i.p.* administration of acetic acid solution. As can be seen on Table 2, extract exhibited significant protection at 100, 200 and 400 mg/kg ($p < 0.001$) with maximum percentage inhibition of constrictions of 80.22% observed at 400 mg/kg while indomethacin (50 mg/kg) had only 83.76% inhibition.

3.3.2 Formalin- induced pain test

The results of this assay are presented in Table 3. There was a significant ($p < 0.01$) reductions in response to nociception during the first and second phases of the pain at all extract doses. Maximum percentage inhibition of nociceptive

Table 1. Qualitative phytochemical composition of the aqueous stem barks extract of *M. lucida*

Triterpenes	Sterols	Flavonoïds	Alkaloïds	Phenols	Saponins	Galic tannins	Catechic tannins
+	-	+	+	+	+	-	+

+: Present, -: Absent

Table 2. Antinociceptive effect of the aqueous stem bark extract of *Morinda lucida* on writhing induced by acetic acid in mice

Groups	Doses (mg/kg)	Number of writhings within 30 mn	Inhibition (%)
Control	/	122.16 ± 16.74	/
Indomethacin	50	19.83 ± 5.90 ***	83.76
<i>M. lucida</i>	100	41.00 ± 2.62 ***	66.43
<i>M. lucida</i>	200	32.00 ± 2.35 ***	73.80
<i>M. lucida</i>	400	24.16 ± 1.72 ***	80.22

Each value represents the mean ± ESM of 6 animals. *** $p < 0,001$ statistically significant compared to control

Table 3. Antinociceptive effect of the aqueous stem bark extract of *Morinda lucida* on formalin-induced pain in mice

Groups	Doses (mg/kg)	First phase (0-5 mn)	Inhibition (%)	Second phase (15-30 mn)	Inhibition (%)
Control	/	267,52 ± 5,82	/	741,24 ± 33,00	/
Indomethacin	50	259,09 ± 14,77	03.15	356,14 ± 33,01***	51.95
<i>M. lucida</i>	100	191,33 ± 23,55 ^{aa}	28.48	338,66 ± 97,20 ***	54.31
<i>M. lucida</i>	200	130,50 ± 11,90 ^{***aaa}	51.22	217,86 ± 44,67 ^{***aa}	70.61
<i>M. lucida</i>	400	124,53 ± 13,15 ^{***aaa}	53.45	178,77 ± 31,30 ^{***aaa}	75.88

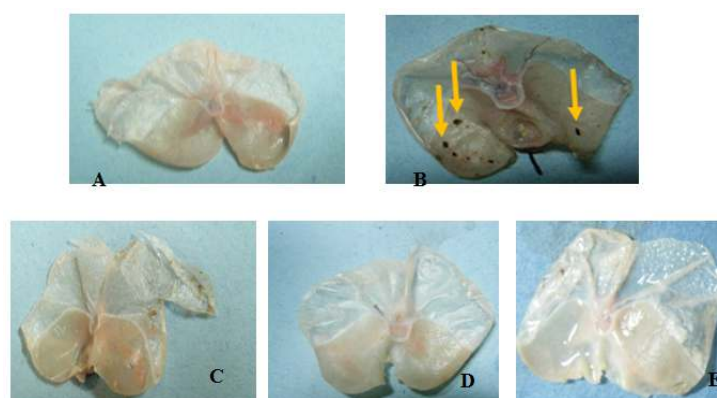
Each value represents the mean ± ESM of 6 animals. $p < 0, 05$ statistically significant compared to control; *** $p < 0,001$ statistically significant compared to control; ^a $p < 0,001$ statistically significant compared to indomethacin; ^{aa} $p < 0,01$ statistically significant compared to indomethacin; ^{aaa} $p < 0,001$ statistically significant compared to indomethacin

effect was 75.88% in the second phase at 400 mg/kg. Indomethacin was significantly active (51.95%, $p < 0.001$) only on second phase. During the two phases, the aqueous stem bark extract of *Morinda lucida* (200 and 400 mg/kg) was significantly ($p < 0.01$) active than indomethacin (50 mg/kg).

3.4 Non-ulcerogenic Properties

Fig. 1 shows the appearance of the mice stomach five hours after the various treatments.

It appears that the distilled water as well as the different doses of extract produced any ulceration in opposition to indomethacin, which caused ulcerations with an ulcer index of 2.5 corresponding to an ulcerated surface area of 2.71 mm². Moreover, unlike indomethacin, which inhibited mucus synthesis, the aqueous stem bark extract of *Morinda lucida* at doses of 200 mg/kg and 400 mg/kg induced significantly ($p < 0.05$) increase in mucus secretion compared to control and indomethacin (Table 4).

**Fig. 1. Macroscopic aspect of the gastric mucosa in mice**

(A) Normal control group. (B) Group treated with indomethacin (50 mg/kg) (ulcer control). (C), (D) and (E) Groups treated with aqueous extract of *Morinda lucida* respectively at 100 mg/kg, 200 mg/kg and 400 mg/kg.

→ : Ulcer indication

Table 4. Non-ulcerogenic effect of the aqueous stem bark extract of *Morinda lucida* on the gastric mucosal in mice

Groups	Doses (mg/kg)	Mucus production (mg)	Ulcerated surface (mm ²)	Ulcer index	% of Ulcerated surface
Control	/	12.16 ± 1.32	0.00	0.00	00
Indomethacin	50	5.00 ± 0.68**	5.54 ± 2,09	2.50 ± 0.60	2.71 ± 1.02
<i>M. lucida</i>	100	14.16 ± 1.19 ^{aa}	0.00	0.00	0.00
<i>M. lucida</i>	200	19.50 ± 3.05 ^{aaa}	0.00	0.00	0.00
<i>M. lucida</i>	400	22.00 ± 0.57 ^{aaa}	0.00	0.00	0.00

Each value represents the mean ± ESM of 6 animals. $p < 0, 05$ statistically significant compared to control; $p < 0, 01$ statistically significant compared to control, ^a $p < 0, 05$ statistically significant compared to indomethacin, ^{aaa} $p < 0,001$ statistically significant compared to indomethacin

4. DISCUSSION

The aqueous stem bark extract of *Morinda lucida* (ASBEML) caused a significant ($p < 0.05$) dose-dependent inhibition of the number of abdominal contractions induced by the intraperitoneal injection of acetic acid (Table 2). Pain induced by acetic acid in mice is described as a typical pattern of inflammatory pain. It is used as an instrument for evaluating the analgesic properties of drogues. Intraperitoneal injection of acetic acid causes pain, either by stimulating chemoreceptors *via* the activation of ASIC (Acid-Sensing Ionic Channel) channels, or by irritating the visceral surface leading to the release of many chemical mediators involved in pain such as histamine, prostaglandins E₂ (PGE₂), serotonin, bradykinin [27]. Indeed, Deraedt et al. [28] found large amounts of PGE₂ and prostaglandins F₂ (PGF₂) in the peritoneal exudate of rats, the first 30 minutes after acetic acid injection. In addition, Ito et al. [29] demonstrated that PGE₂ induces acute cramps in response to noxious chemical stimuli. ASBEML could thus act either by blocking the ASIC channels or by inhibiting the synthesis of prostaglandins and other mediators of pain [30]. Acetic acid also induces the synthesis of chemical mediators at the central level, such as substance P. Also, the anti-nociceptive properties of opioids, partial agonists of opioids and nonsteroidal anti-inflammatory drugs can be determined by this method. The results of this test do not make it possible to say with precision in which of these three classes lies this extract. Since acetic acid simulates both the effects of peripheral and central pain, this method is insufficient to locate the site of action of ASBEML.

It was therefore with the aim of locating the site of action of ASBEML that a second test was carried out, namely the induction of pain by

formaldehyde. Formaldehyde has the advantage of separating the components of peripheral pain from central pain [31]. Injecting a solution of formaldehyde into the mouse causes a diphasic response. The first phase, called neurogenic, is triggered immediately after the injection of the formaldehyde solution. It is characterized by the release of substance P and stimulation of the vanilloid receptors with transmission of the nerve message by the C fibers. The second phase, called the inflammatory or peripheral phase, is due to the local inflammatory pain caused by the production of serotonin, bradykinin, histamine and prostaglandins [32]. Central analgesics such as opioids inhibit both phases of this test while peripheral analgesics (NSAIDs) act on the peripheral phase [33]. The second phase of this test is used to determine the anti-hyperalgesic effects of certain substances on models of neuropathic pain [34]. The present study shows that ASBEML significantly ($p < 0.05$) reduced the two phases of pain induced by formaldehyde injection with more pronounced action in the second phase (Table 3). This suggests that the analgesic effect of ASBEML may be due to inhibition of substance P synthesis and / or blockade of vanilloid receptors. This analgesic effect would also be due to the inhibitory action of ASBEML on the synthesis of proinflammatory mediators (bradykinin, serotonin, histamine and prostaglandins) or inhibition of the nociceptive effect of neurotransmitters (glutamate) which act as a facilitator descending from pain [35]. The presence of flavonoids and tannins in ASBEML [36] may be at the origin of the observed analgesic effect. Flavonoids and tannins have been shown to have potent analgesic effects [37]. Indeed, flavonoids are inhibitors of the synthesis of prostaglandins, powerful mediators of pain [38]. The pronounced action of ASBEML on the second phase of formaldehyde-induced pain and on pain induced by acetic acid suggests that the ASBEML acts *via* prostaglandins like

NSAIDs. NSAIDs (indomethacin) cause peptic ulcers in patients. It was therefore necessary to carry out an ulcerogenic test to verify whether ASBEML has this side effect.

There are two types of cyclooxygenase (COX) in the body: cyclooxygenase 1 (COX1) and cyclooxygenase 2 (COX2). COX 2, known as inducible, is responsible for the synthesis of prostaglandins during inflammatory reactions. COX 1 is a constitutional enzyme that governs the synthesis of prostaglandins involved in gastric cytoprotection. NSAIDs such as indomethacin act by inhibiting COX 1 and 2. This results in an inhibition of the synthesis of prostaglandins, a potent mediator of pain. However, prostaglandins play an important cytoprotective role in the stomach. They stimulate the secretion of mucus and HCO₃⁻ bicarbonate ions which protect the gastric mucosa [7]. The inhibition of prostaglandin synthesis by NSAIDs is directly correlated with the onset of gastric ulcers, as shown by the ulcerogenic test (Table 4). ASBEML at all doses did not cause gastric ulcer. On the contrary, this extract stimulated mucus secretion (Table 4). This suggests that ASBEML acts by selectively inhibiting COX 2. The increase in mucus secretion may be due to the stimulation of prostaglandin synthesis *via* COX1 or by direct stimulation of production of mucus by triterpenes [39]. The presence of triterpenes in ASBEML, as revealed by Addy et al. [35], was confirmed by phytochemical tests (Table 1). This extract of *Morinda lucida* could therefore also have anti-ulcerogenic effects.

In vitro, Folin test revealed that ASBEML had a total polyphenol content of 299.89 ± 1.62 mg Ascorbic acid / g of dry extract. This result suggests that this extract is rich in polyphenols. Using the DPPH method, this extract exhibited an antiradical activity of 49.44%. ASBEML shows a slightly higher antiradical activity than the aqueous stem bark extract of *Alstonia boonei* (41.58%) [40]. This antioxidant activity is linked to the richness of the polyphenol extract. The polyphenolic compounds have an antioxidant activity linked to the redox properties which enable them to act as reducing agents, metal chelators and as free radical scavengers [41]. In addition, Sokol-Letowska's study shows that phenolic compounds (flavonoids, tannins) possess significant antioxidant activity [42]. This is due to the presence of many hydroxide groups that can react with free radicals. This antioxidant activity is confirmed by the FRAP test which

reveals an antioxidant potential of ASBEML of 994.53 ± 6.11 mg Ascorbic acid / g of dry extract, ie 99453 mg of ascorbic acid / 100 g of dry extract. Most effective medicinal plants are rich in polyphenols and possess high antioxidant potential [40]. In addition, ASBEML leads to an increase in the concentration of super oxide dismutase (SOD) and catalase, and a decrease in malondialdehyde (MDA) in diabetic rats [14].

5. CONCLUSION

The aqueous stem bark extract of *Morinda lucida* possesses analgesic and antioxidant properties, and has anti-ulcerogenic properties. This would justify the use of this plant in traditional medicine.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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