Investigating the Site of Action of an Aqueous Extract of *Heliotropium indicum* Linn (Boraginaceae) on Smooth Muscles

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**Abstract:** *Heliotropium indicum* has various traditional medicinal uses such as treating abdominal pains, dysmenorrhea, hypertension, convulsion, post-partum inflammatory disorders, wounds and infections and skin rashes. The aim of this study therefore is to find receptors that possibly mediate the activity of *H. indicum* as a means of finding explanation to some of its reported traditional uses. The effect of various concentrations of agonist drugs and an aqueous extract from the plant as well as the effects of these agonist drugs and the extract in the presence of specific reference antagonist drugs were established on isolated guinea-pig ileum, rabbit jejunum, rat uterus and rat anococcygeus preparations. Data obtained was analyzed using GraphPad Prism Version 5.0 for Windows. The extract caused dose-dependent contractions similar to the acetylcholine, methylecholine, carbamylcholine, nicotine, histamine and oxytocin used on the smooth muscle preparations studied. The contractions were significantly inhibited by atropine and hexamethonium, suggesting muscarinic and nicotinic activity, adrenaline and salbutamol, suggesting adrenoceptor activity and diclofenac sodium, suggesting the inhibition of synthesis and/or effect of products of COX as prostaglandin. The extract was significantly stable to plasma cholinesterase. The receptor activity of *H. indicum* explains some of its traditional medicinal uses such as relieving abdominal pain, hypertension and impotence and sexual weakness.

**Key words:** Cholinesterase stability, hexamethonium, muscarinic activity, nicotinic activity, carbamylcholine, COX

**INTRODUCTION**

*Heliotropium indicum* is an annual herb commonly known as the Cocks comb. It is usually associated with the moist rich soils of the lowland tropics near rivers and lakes on the road sides and also in waste places (Holm et al., 1977). In Ghana, a cold infusion of the leaves has been used as an enema to stop abdominal pains and treat catarract; the juice from the leaves is squeezed into the eye to stop dizziness; decoction of the whole plant is used to treat convulsion in children; the roasted aerial parts of the plant together with certain ingredients are used as an enema for expulsion of clotted blood in a women who have recently given birth, the poultice of the leaves mixed with honey is externally applied to the perine to restore virility (Irvine, 1961), the juice from the mashed leaves is used as ear drops (Burkill, 1985) and the poultice is topically applied to swollen glands of the neck. Irvine (1961) had reported the use of the concoction of the leaves with clay as an anti-abortive agent among Asante women. Previous researches on this plant includes wound healing activity of *H. indicum* (Reddy et al., 2002; Diwan et al., 1982; Udupa et al., 1989) had reported that chloroform extract of *H. indicum* dose-dependently inhibited the carrageenan induced rat paw oedema and also showed anti-nociceptive activity in rats. Kugelman et al. (1976) isolated N-oxide of the alkaloid indicine from *H. indicum* and observed that it has significant anti-tumor activity. Crude hexane extract of *H. indicum* had antimicrobial activity against *Mycobacterium tuberculosis* (H37Ra) (Machinian et al., 2005). The extensive medicinal use of *H. indicum* is not paralleled by adequate scientific data there is therefore, the need to add to the existing scientific data to provide
some pharmacological evidence for its multi-medicinal use, *H. indicum* extract is being investigated on isolated smooth muscle preparations.

**MATERIALS AND METHODS**

**Plant collection:** The aerial parts of *Heliotropium indicum* were collected from Buokrom, a suburb of the Ashanti region of Ghana in September, 2007 and authenticated by the Curator (Mr. Adator K. Brown) of the Department of Theoretical and Applied Biology, College of Sciences, KNUST where a voucher specimen (KNUST/BSC/F621) was deposited.

**Preparation of extract:** A 600 g quantity of the coarse powder was mixed with 5 L of water and warmed for 15 min. The infusion was filtered to obtain a dark-brown filtrate, concentrated by evaporation over a hot water bath and later in a hot air-oven at 60°C until a constant weight was obtained. It was cooled in a desiccator to yield dark-brown solid extract which was named HIE.

**Drugs and chemicals:** Acetylcholine, nicotine, histamine, atropine, hexamethonium, mepyramine, methylcholine, carbamylcholine, phystostigmine, noradrenaline and phenolamine were obtained from Sigma-Aldrich Inc., St. Louis, MO, USA. Oxytocin was obtained from Alliance Pharmaceuticals Ltd., UK and Salbutamol from Ernest Chemist, Accra, Ghana. These drugs were agonists and antagonists used in the study.

**Experimental animals:** Mature Sprague-Dawley rats, guinea-pigs and New Zealand White rabbits obtained from the animal house of the Department of Pharmacology, KNUST were used in this study. They animals were kept under ambient environmental conditions and had access to normal rat, guinea-pig and rabbit chow and water *ad libitum*.

**Phytochemical evaluation:** HIE was screened for phytochemical using standard techniques of plant metabolites as described by Sofowora (1993), Harborne (1998) and Trease and Evans (1989).

**Isolated guinea-pig ileum:** A guinea-pig was sacrificed and the ileum isolated as per the method described by Okpako and Taiwo (1984). It was mounted in Tyrode physiological solution maintained at 32°C with constant aeration using Corning-Bel, 850 air compressor (Evans Electrooselenium Ltd., Halstead Essex England) in a Harvard tissue bath (Harvard Apparatus Ltd., Kent, UK). The baseline response was recorded by means of a pendular lever system with the frontal writing point moving on a white paper wound around a 30 cm diameter cylinder of Harvard kymograph (Harvard Apparatus Ltd., Kent, UK) revolving at a rate of 4 mm min⁻¹. Acetylcholine (1×10⁻⁵-2.6×10⁻³ mg mL⁻¹), histamine (1×10⁻³-2.6×10⁻¹ mg mL⁻¹), nicotine (1×10⁻⁴-2.6×10⁻² mg mL⁻¹) and HIE (2×10⁻⁵-5.12 mg mL⁻¹) were applied on the guinea-pig ileum and the contractile responses recorded using 3 min time cycle and a 30 sec contact time. The procedure was repeated four times.

**Determination of site of action of HIE on the guinea-pig ileum:** A complete dose-response tracings of acetylcholine (8×10⁻⁵-1.3×10⁻³ mg mL⁻¹) was established using the isolated guinea-pig ileum. A 70-75% sub-maximal response produced by acetylcholine (6×10⁻³ mg mL⁻¹) was selected. Equipotent responses to the sub-maximal response of acetylcholine produced by nicotine (4×10⁻⁴ mg mL⁻¹), histamine (3.2×10⁻⁴ mg mL⁻¹) and HIE (2.7 mg mL⁻¹) were established and matched. Attempts were made to establish the equipotent responses again in the presence of hexamethonium (0.005 mg mL⁻¹), atropine (1×10⁻⁴ mg mL⁻¹), mepyramine (2×10⁻² mg mL⁻¹) and contractile responses recorded and compared. The procedure was repeated 4 times.

**Test for stability of HIE to plasma cholinesterase:** A complete dose-response tracings of acetylcholine was established and a sub-maximal response (70% of the maximum response) given by 1×10⁻⁴ mg mL⁻¹ was chosen. Equipotent responses for methylcholine (1×10⁻⁴ mg mL⁻¹) carbamylcholine (3×10⁻⁴ mg mL⁻¹) and HIE (8.5 mg mL⁻¹) were established. Ten test tubes were labelled I-X and treated as shown in Table 1.

While 10-fold sub-maximal dose of acetylcholine and equipotent doses of methylcholine, carbamylcholine and HIE were added to test tubes I-IV, respectively and made up with distilled water up to 10 mL to serve as controls. To the tenfold dose of acetylcholine (test tube V), methylcholine (test tube VI), carbamylcholine (test tube VII) and the HIE (test tube VIII) was added 3 drops of blood (source of cholinesterase) and then made up to 10 mL with distilled water.

To test tube IX (control), 3 drops of blood and 5 mL distilled water were added, boiled for 5 min and cooled before the addition of the 10-fold dose of acetylcholine. It was then made up with distilled water to 10 mL. To confirm that cholinesterase was responsible for the expected inhibitions, 3 drops of blood and 1 mL of phystostigmine (5×10⁻⁵ mg mL⁻¹) was added to test tube X before the addition of the 10-fold dose of acetylcholine. It was then made up with distilled water to 10 mL. The test
Table 1: Treatment of test tubes I-X in the determination of stability of acetylcholine, methlycholine, carbachol and HIE to cholinesterase activity

<table>
<thead>
<tr>
<th>Test tubes</th>
<th>I</th>
<th>II</th>
<th>III</th>
<th>IV</th>
<th>V</th>
<th>VI</th>
<th>VII</th>
<th>VIII</th>
<th>IX</th>
<th>X</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acetylcholine</td>
<td>0.2 mL</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>0.2 mL</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>0.2 mL</td>
<td>0.2 mL</td>
</tr>
<tr>
<td>Methlycholine</td>
<td>-</td>
<td>0.5 mL</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Carbachol</td>
<td>-</td>
<td>-</td>
<td>0.5 mL</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>HIE</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>0.5 mL</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Blood</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>3 drops</td>
<td>3 drops</td>
<td>3 drops</td>
<td>3 drops</td>
<td>3 drops*</td>
<td>3 drops</td>
</tr>
<tr>
<td>Physostigmine</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>1 mL</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Distilled water</td>
<td>10 mL</td>
<td>10 mL</td>
<td>10 mL</td>
<td>10 mL</td>
<td>10 mL</td>
<td>10 mL</td>
<td>10 mL</td>
<td>10 mL</td>
<td>10 mL</td>
<td>10 mL</td>
</tr>
</tbody>
</table>

*The blood was added to distilled water, boiled and cooled before adding 10 volume of acetylcholine that yielded the sub-maximal dose.

tubes were incubated for 15 min in water bath at 37°C which is the optimum temperature for cholinesterase activity. After the incubation period, 1 mL of each drug equivalent to the selected dose in the respective test tubes were administered onto the mounted isolated guinea-pig ileum preparation in succession and the dose-response tracings obtained were analyzed. The procedure was repeated three times.

Isolated rabbit jejunum: A rabbit was sacrificed and the jejunum isolated as earlier described by Okpako and Taiwo (1984). It was mounted in a Harvard tissue bath in Ringer Locke physiological maintained at 37°C with constant aeration. The baseline response was recorded by means of a pendular lever system with the frontal writing point moving on a white paper wound around a 30 cm diameter cylinder of Harvard kymograph revolving at a rate of 8 mm min⁻¹. The contractile effects of acetylcholine (1×10⁻⁷-3.2×10⁻² mg mL⁻¹), nicotine (1.25×10⁻² mg mL⁻¹) and HIE (3×10⁻⁷-34 mg mL⁻¹) were established and the reestablished in the presence of atropine (8.3×10⁻² mg mL⁻¹) and hexamethonium (0.01 mg mL⁻¹). A time cycle of 3 min and a contact time of 30 sec was used. The procedure was repeated four times.

Isolated rat uterus: The two horns of the uterus of a female Sprague-Dawley rat were isolated and mounted in a Harvard tissue bath in De Jalon’s physiological solution maintained at 32°C as previously described by Okpako and Taiwo (1984). The two horns were separated and freed from fat and connective tissues and each was cut open longitudinally. A strip of the horn (about 2-3 cm) was cut out. One end of the isolated uterine strip and mounted on the tissue holder in the organ bath containing De Jalon’s physiological solution (maintained at 32°C and constantly aerated) with a piece of cotton thread while the other end was attached to a pendular lever system with the frontal writing point moving on a white paper wound around a 30 cm diameter cylinder of Harvard kymograph revolving at a rate of 4 mm min⁻¹.

After 1 h, the baseline response was recorded. Responses of oxytocin (1×10⁻²-6.67×10⁻² mg mL⁻¹), acetylcholine (1×10⁻²-3.2×10⁻² mg mL⁻¹) and HIE (1.3×10⁻²-17.1 mg mL⁻¹) applied on the tissue preparation and these in the presence of reference antagonists drugs adrenaline (1×10⁻⁷ mg mL⁻¹), salbutamol (4.2×10⁻⁷ mg mL⁻¹), atropine (4.2×10⁻⁸ mg mL⁻¹) and diazepam sodium (1.7×10⁻⁴ mg mL⁻¹). The contractile responses recorded using 3 min time cycle and a 30 sec contact time. The procedure was repeated 4 times.

Isolated rat anococcyeus: This method was previously described by Gillespie (1972) with some modification. The abdomen of a male rat was opened in the mid-line, the pelvis split and the bladder and urethra removed. Care was taken in clearing the lower part of the urethra to avoid damage to the ventral bar of the muscle, the only region lying ventral to the colon. The colon was then cut through at the pelvic brim, the portion pulled forward and the delicate connective tissue behind cleared until the anococcyeus muscles came into view. The muscle was isolated with the extrinsic nerve intact. The ventral bar was cut through and each muscle mounted in a 20 mL tissue bath containing Kreb’s solution at 32°C. The tissue was aerated with 95% O₂+5% CO₂. The anococcyeus was tied anteriorly to a hook in the tissue bath while the posterior end was tied to a lever system with a writing pointer on a white kymograph paper wound around a drum revolving at a rate of 4 mm min⁻¹. Base line tracings were obtained. The responses of noradrenaline (2×10⁻⁷-2×10⁻⁵ mg mL⁻¹), acetylcholine (4×10⁻⁷-4×10⁻⁵ mg mL⁻¹) and HIE (1-32 mg mL⁻¹) and these in the presence of phentolamine (1×10⁻⁶ mg mL⁻¹) and atropine (4×10⁻⁶ mg mL⁻¹) were recorded.

Statistical analysis: GraphPad Prism Version 5.0 for Windows (GraphPad Software, San Diego, CA, USA) was used for all statistical analyses. Data are presented as mean±SEM and analyzed by one-way ANOVA followed by Bonferroni’s multiple comparisons test (post test). The p<0.05 was considered statistically significant in all analysis.

RESULTS AND DISCUSSION

Phytochemical screening: Phytochemical screening of HIE showed the presence of alkaloids, cyanogenic glycosides, tannins, saponins and steroids.
Fig. 1: Log-dose response curves of acetylcholine, histamine and nicotine at doses of 1×10⁻⁴-2.6×10⁻³ mg mL⁻¹ and HIE (2×10⁻⁵-5.12 mg mL⁻¹) on the isolated guinea-pig ileum preparation. Each point represents the mean±SEM (n = 4).

Table 2: The inhibitory effect (%) of atropine, hexamethonium and mepivacaine on the contractile activity of acetylcholine, nicotine, histamine and HIE.

<table>
<thead>
<tr>
<th>Antagonist</th>
<th>Acetylcholine</th>
<th>Nicotine</th>
<th>Histamine</th>
<th>HIE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atropine</td>
<td>7.2±0.959</td>
<td>3.0±0.5774</td>
<td>3.0±0.9125</td>
<td>34.8±0.1999</td>
</tr>
<tr>
<td>Hexamethonium</td>
<td>2.3±0.853</td>
<td>8.6±0.9950</td>
<td>1.2±0.750</td>
<td>38.9±1.5900</td>
</tr>
<tr>
<td>Mepivacaine</td>
<td>2.3±0.6292</td>
<td>3.8±0.853</td>
<td>65.7±0.1150</td>
<td>0.0±0.0000</td>
</tr>
</tbody>
</table>

Values are mean±SEM (N = 4).

**Effect of HIE on the isolated guinea-pig ileum:** HIE produced dose-dependent contractions of the isolated guinea-pig ileum comparable to acetylcholine, histamine and nicotine (Fig. 1). The contractile effects were inhibited by atropine and hexamethonium but not mepivacaine. The percentage inhibitions are as shown in Table 2. HIE showed stability to plasma cholinesterase comparable to those of carbamylcholine and methylcholine (Fig. 2).

**Effect of HIE on the rabbit jejunum:** HIE produced biphasic effect on the jejunum; relaxing the rhythmic contractions of the jejunum at lower concentrations and dose-dependent contractions at higher doses (0.3-34.1 mg mL⁻¹). The effects of HIE in the contractile phase was similar to effects of acetylcholine and nicotine. Atropine and hexamethonium inhibited the contractile effects of HIE (Fig. 3).

**Effect of HIE on the isolated rat uterus:** HIE produced dose-dependent myometrial contractions on the isolated rat uterus preparations similar to those produced by acetylcholine and oxytocin (Fig. 4). The myometrial contractions caused by HIE and acetylcholine were inhibited by atropine. These drugs were physiologically antagonized by adrenaline (Fig. 5). The myometrial contractions caused by HIE and oxytocin were inhibited by salbutamol but the myometrial contractions caused by HIE (but not oxytocin) was inhibited by diclofenac sodium (Fig. 6).

**Effect of HIE on the isolated rat anococoegeus muscle:** HIE produced a concentration-dependent contraction of the isolated rat anococoegeus muscle and these contractions were competitively inhibited by atropine and phenolamine (seen as a parallel shift of the curve to the
Fig. 4: Log-dose response curves of oxytocin (8.3×10^{-5}-5.3×10^{-3} IU mL^{-1}), acetylcholine (1×10^{-2}-3.2×10^{-2} mg mL^{-1}) and HIE (0.13-17.1 mg mL^{-1}) on the isolated non-pregnant rat uterus. Each point is the mean±SEM (n = 5).

Fig. 5: Inhibitions of the myometrial contractions of HIE (1.3×10^{-1}-17.1 mg mL^{-1}) and acetylcholine (1.3×10^{-1}-17.1 mg mL^{-1}) by atropine (4.2×10^{-4} mg mL^{-1}) and adrenaline (1×10^{-3} mg mL^{-1}) on the isolated rat uterus. Each column is the mean±SEM (n = 4). Inhibition of responses: HIE by atropine, **p<0.01; acetylcholine by atropine, *p<0.05, HIE by adrenaline, ***p<0.001, acetylcholine by adrenaline, ***p<0.001.

Fig. 6: Inhibitions of the myometrial contractions of HIE (1.3×10^{-3}-17.1 mg mL^{-1}) and oxytocin (1×10^{-9}-6.67×10^{-7} mg mL^{-1}) by salbutamol (4.2×10^{-4} mg mL^{-1}) and diclofenac sodium (1.7×10^{-4} mg mL^{-1}) on the isolated rat uterus. Each column is the mean±SEM (n = 4). Inhibition of responses: HIE by salbutamol, **p<0.01; oxytocin by salbutamol, **p<0.01, HIE by diclofenac, *p<0.05, oxytocin by diclofenac, ns; p>0.05.

Fig. 7: The contractile effects of acetylcholine (4×10^{-7}-4×10^{-5} mg mL^{-1}) and HIE (1-32 mg mL^{-1}) in the absence of and in presence of atropine (4×10^{-6} mg mL^{-1}). The parallel shift of the acetylcholine and HIE curves to the right indicates the presence of atropine indicates competitive inhibition.

right). Acetylcholine and noradrenaline also produced contractile responses that were also competitively inhibited by atropine and phentolamine, respectively (Fig. 7 and 8). The purpose of this study was to provide pharmacological bases and possible mechanism(s) of action of an aqueous extract from *H. indicum* on isolated smooth muscles, thus supporting some of it traditional uses in Ghana. A repertoire of tissues was employed in the study in order to assess as many receptors as possible as well as providing direct evidence to the ethnopharmacological use of the plant. The isolated guinea-pig ileum is a low tone tissue which has muscarinic (Giraldo *et al*., 1988; Bolton and Zholos, 1997), histaminic (Bertaccini *et al*., 1979; Barker and Ebersole, 1982), opioid (Campbell *et al*., 1989) and neurokinin (Nguyen-Le *et al*., 1996) receptors. Because the autonomic ganglion (which has nicotinic receptors) is so close to the tissue, an isolated ileum will have nicotinic receptor activity.
sympathetic nerves causes noradrenergically mediated contractions (Gillespie, 1972). Contractions of the rat anococcygeus muscle elicited by acetylcholine can be attributed to activation of postjunctional muscarinic receptors (Gillespie, 1972). On the isolated rat anococcygeus muscle, HIE produced concentration-dependent contractile effect which was competitive inhibited by atropine and phenotolamine (a non-selective \(\alpha\)-adrenoceptor antagonist) suggesting that the extract may have chemical constituents with muscarinic and possibly \(\alpha\)-adrenoceptor agonist activity.

To lend some information to support or prove otherwise the traditional uses of the plant in conditions involving the uterus assessment of HIE on the isolated rat uterus was conducted. The isolated rat uterus has among a number muscarinic (Choppin et al., 1999), adrenergic (Paton, 1968) and oxytocic (Engstrom et al., 2000) receptors. The assessment of HIE on the isolated rat uterus was conducted to lend some information to support or prove otherwise the traditional uses of the plant in conditions involving uterus.

The extract produced concentration-dependently myometrial contraction just as acetylcholine and oxytocin and these contractions were inhibited by atropine, adrenaline (non-selective adrenoceptor agonist), salbutamol (\(\beta_2\)-adrenoceptor agonist) and diclofenac (cyclooxygenase enzyme inhibitor). These suggest that the contractile effect of HIE may be mediated via a cholinergic mechanism, a blockade of adrenoceptors (possibly \(\beta_2\)-adrenoceptors) and possibly through the enhancement of prostaglandin synthesis (Monnneau et al., 1984). One traditional use of \textit{H. indicum} is the relief of abdominal pain. One cause of abdominal pain is the inability to empty the filled urinary bladder possible due to a malfunctioning urinary bladder due to neurologic dysfunction or insult emanating from internal or external trauma, disease or injury termed neurogenic bladder. The muscarinic activity suggests that the plant extract of can be used to assist bladder emptying (Chess-Williams, 2002).

Furthermore, the use of HIE for abdominal pain may stem from its ability to activate the \(\alpha\)-adrenoceptors which in effect lead to decreased motility and spasms (source of pain such as abdominal cramps) of the GIT as well as visceral organs. More so, the leaves of \textit{H. indicum} can serve as home remedy for poisoning arising from pesticides such as D-tubocurarine that act by depleting ACh from the neuromuscular junction. Activation of the parasympathetic nervous system innervating the penis produces cGMP and thus gives rise to an erection (Kandeel et al., 2001). Muscarinic-receptor stimulation by
acetylcholine increases arterial flow, cavernous smooth muscle relaxation and venous occlusion (Stief et al., 1989). The stimulation of spinal cord acetylcholine receptors has been reported to facilitate ejaculation (Gutman and Walsh, 1970; Chapelle et al., 1993; Vargas et al., 2004). Induction of prostaglandin synthesis causes vasodilatation of penile arteries which enhances erection (Minhas et al., 2001). This effect may support its traditional use of externally applying to the penis to restore virility (Irvine, 1960). Muscarinic activity on the myocardium which results in a negative inotropic and chronotropic effect could contribute immensely to reducing blood pressure; the product of cardiac output and total peripheral resistance. By the negative effect, cardiac output will be reduced. This could lend an explanation to the use of the plant as an antihypertensive. The muscarinic agonist activity and enhanced synthesis/release of prostaglandin of HIE augmented by its anti-sympathetic activity on β1-receptors may again support its use as an enema for expulsion of clotted blood in women who have recently given birth. Clots of blood in the uterus after childbirth (post-labour disorder) may also result in abdominal pain. Further, in vitro research is needed to provide a direct and clearer mechanism of action of the contractile effects of the extract on the uterus. Thus, the use of plant in pregnant women must be done with caution. The use of H. indicum in dysmenorrhea, post-partum inflammatory disorders, wounds and infections and skin rashes could be due to its anti-inflammatory (Srinivas et al., 2000) and antimicrobial activity (Dash and Murthy, 2011) which it has been reported. Phytochemical identified are in conformity to that found in previous studies (Zhao et al., 1989).

CONCLUSION

In the study, the extract of H. indicum has chemical constituents that have stimulatory effects at the muscarinic and nicotinic receptors, α and β-adrenergic receptors and possible enhance prostaglandin synthesis. These effects explain some of its reported traditional uses in Ghana although, further studies are recommended to establish its safety for use.

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