Effects of aqueous leaf extract of *Asystasia gangetica* on the blood pressure and heart rate in male spontaneously hypertensive Wistar rats

Pierre Mugabo1* and Ismaila A Raji2

Abstract

**Background:** *Asystasia gangentica* (*A. gangetica*) belongs to the family Acanthaceae. It is used to treat hypertension, rheumatism, asthma, diabetes mellitus, and as an anthelmintic in South Africa, India, Cameroun, Nigeria, and Kenya respectively. It has also been reported to inhibit the angiotensin I converting enzyme (ACE) *in-vitro*. Therefore, the aim of this study is to investigate the *in-vivo* effect of aqueous leaf extract (ALE) of *A. gangetica* on the blood pressure (BP) and heart rate (HR) in anaesthetized male spontaneously hypertensive rats (SHR); and to elucidate possible mechanism(s) by which it acts.

**Methods:** The ALE of *A. gangetica* (10–400 mg/kg), angiotensin I human acetate salt hydrate (ANG I, 3.1–100 μg/kg) and angiotensin II human (ANG II, 3.1–50 μg/kg) were administered intravenously. The BP and HR were measured via a pressure transducer connecting the femoral artery to a Powerlab and a computer for recording.

**Results:** *A. gangetica* significantly (p<0.05), and dose-dependently reduced the systolic, diastolic, and mean arterial BP. The significant (p<0.05) reductions in HR were not dose-dependent. Both ANG I and ANG II increased the BP dose-dependently. Co-infusion of *A. gangetica* (200 mg/kg) with either ANG I or ANG II significantly (p<0.05) suppressed the hypertensive effect of both ANG I and ANG II respectively, and was associated with reductions in HR.

**Conclusions:** *A. gangetica* ALE reduced BP and HR in the SHR. The reduction in BP may be a result of actions of the ALE on the ACE, the ANG II receptors and the heart rate.

**Keywords:** *Asystasia gangetica*, Blood pressure, Heart rate, Spontaneously hypertensive rats, Renin angiotensin aldosterone system

Background

A great number of plants have been used throughout the world from time immemorial for the prevention and cure of sickness [1]. About eighty percent of the active compounds of drugs used for the treatment of cardiovascular conditions are extracted from medicinal plants, e.g. foxglove (*digitalis lanata*), *Allium sativum* (garlic), camphor and *Adonis vernalis* [1-3].

Drugs used for the treatment of hypertension include angiotensin converting enzyme inhibitors (ACEIs), α1 adrenoceptor blockers, calcium channel blockers, angiotensin II (ANG II) receptor antagonists, diuretics and beta adrenoceptor blockers [4-7]. Most of these drugs are expensive, and in many cases, none of them can control hypertension on its own. Consequently, combination therapy is often required [5,6]. However, combination therapy (polytherapy) has its drawbacks, such as increased risk of drug interactions, side effects and poor compliance [8-10]. These necessitate continuous search for novel agents which are more effective, cheaper, come with less side-effects, and preferably used alone.

*Asystasia gangentica* (*A. gangetica*) belongs to the family Acanthaceae [11,12]. There are eight species native to Southern Africa, and notably, the KwaZulu Natal province of South Africa [12]. *A. gangetica* is used to treat asthma in Nigeria [13], as an anthelmintic in Kenya...
[14], and as stomachic, astringent, and diaphoretic in India [15]. It has also been suggested to exhibit antihypertensive effects through inhibition of ACE in-vitro [12]. Therefore, the aim of this study was to evaluate the effect of ALE of *A. gangetica* on the blood pressure (BP) and heart rate (HR) in spontaneously hypertensive rats (SHR), and also investigate if inhibition of the ACE or the ANG II receptor mediates its effect in-vivo.

**Methods**

**Study design**
The study was designed as an in-vivo experimental model assessing the effects of *A. gangetica* on BP and HR in male SHR.

**Collection and preparation of plant material**
*A. gangetica* plants were obtained from Newplant nursery in George, Western Cape, South Africa (SA), in March 2009. A sample of the plant was deposited at the University of the Western Cape (UWC) herbarium for identification and authentication by the taxonomist. It was registered under the voucher number 3469. Fresh leaves (1314.27 g) were picked from the plant and washed twice with distilled water. Leaves were permitted to dry completely in a room where the temperature was maintained at 23°C for a period of 14 days. Dried leaves (967 g) were then pulverized to a fine powder (738 g) using a Hammer mill and stored in air-tight glass containers.

**Aqueous extraction of plant material**
A Soxhlet apparatus was used to extract the active compounds from 738 g powder over 48 hours. A Soxhlet thimble was constructed with glass fibre. Fine powder of the *A. gangetica* was wetted with distilled water, and placed between two layers of glass fibre (used as a barrier to prevent powder from passing into the extract). Distilled water (400 ml) was placed in a round bottom flask and connected together along with the reflux condenser. A heating element was used to supply sufficient heat to boil the distilled water. After extraction, the remnant of the crude plant material was discarded. The aqueous extract was then placed in a deep freezer for a period of 72 hours and transferred to a freeze drier for a period of 72 hours and transferred to a freeze drier for 96 hours to produce a dry powder ready for reconstitution and administration. A powder weighing 179 g was obtained.

**Materials and equipment used in the extraction process of the plant**
The materials and equipment used for plant extraction are of standard analytical grade, and include an oven, scissors, weighing balance, glass fibre, Soxhlet extractor, rotovapour (Bach Rotavapor R200, Switzerland), freeze-drier (Virtis FreezeMobile 12SL, SA), 0.45 μm filter paper (Schleicher & Schuell MicroScience, SA) and a −85°C freezer (Snijder Scientific, SA).

**Animals**
Healthy male SHR weighing 250–400 g and aged less than 4 months old were used. The SHR were obtained from the Animal Unit of the University of Cape Town, Cape Town (CPT), SA; housed in the animal room of the School of Pharmacy, UWC; and allowed feed and water ad-libitum. The animal room temperature was kept at 24°C, with a 12:12 h light–dark cycle.

**Materials and equipment used in the in-vivo experiments**
BP transducer (AD Instruments, CPT, SA), BP amplifier (ML117 AD Instruments, CPT, SA), PowerLab 4/20 T (AD Instruments, Lassec, CPT, SA), computer desktop unit, temperature probe (AD Instruments, Lassec, CPT, SA), Chart 5.0 for Windows software (AD Instruments, Lassec, CPT, SA), Ascor AP 22 syringe pump (United Scientific, CPT, SA), heated rat operating table (Bio-Science, CPT, SA), overhead lamp, bulldog clamps, 0.5 mm (arterial and venous) cannula, three-way tap, syringe, threads, oxygen mask, surgical scissors and cotton wool.

**Chemicals and drugs used in the in-vivo experiments**
Sodium chloride (Adcock Ingram, SA), dimethyl sulfoxide (DMSO) (Merck Chemicals, SA), 0.1% heparin sodium (1000 iu/ml, Intramed, SA), 6% sodium pentobarbitone (Kyon Laboratories, Johannesburg, SA). 0.9% Sodium chloride [16] and/or DMSO [17], were respectively used as vehicle to obtain homogenous dilution of *A. gangetica*, heparin, and the unknown drug. All drugs, except where otherwise indicated, were purchased from Sigma Aldrich, SA. ANG I and ANG II which have potential antagonistic effect to that of *A. gangetica*, were used as negative controls of *A. gangetica* [6,7,18]. Fresh solutions were made at the beginning of each experiment. Chemicals, after having been diluted were kept in ice during the course of the experiment to keep them stable.

**Animal preparation and recording of experimental parameters assessed**
After the induction of anesthesia with sodium pentobarbitone (40 mg/kg) intraperitoneally, the rat was placed in a supine position on a heated operating table to maintain the temperature of the body temperature at ±37°C. A temperature probe was inserted into the rectum and used to measure the core temperature via the amplifier connected to the PowerLab® and monitored with a computer throughout the experiment. Tracheotomy was performed and a tube placed in the trachea for easy breathing. Rats were supplied with oxygen through a
facial-oxygen mask to keep the air environment surrounding the tracheal cannula rich in oxygen. The external jugular vein and the femoral artery were cannulated with catheters containing normal saline/heparin solution in the ratio of 9 ml to 1 ml, for the intravenous infusion of known drugs and/or the ALE of *A. gangetica*. In experiments where *A. gangetica* was co-administered with a control drug, both external jugular veins were cannulated for simultaneous intravenous administration. One of the femoral arteries was cannulated for measurement of arterial BP [19]. BP was measured as systolic pressure (SBP), diastolic pressure (DBP), and mean arterial pressure (MAP). Both BP and HR were monitored continuously on a computer running the Chart 5 software (AD Instruments, Lasec CPT, SA) through a BP transducer linking the arterial cannula to a PowerLab® via a BP amplifier. Randomized doses of drugs and extract were administered during the study.

**Dissolution and infusion of drugs**

All drugs and plant extract were dissolved in 0.9% NS and administered per rat body weight in a volume not higher than 0.5 ml per dose. DMSO (2 to 3 drops) was added to the plant extracts for homogenous dilution and filtered before administration. Drugs were infused at a rate based on the body weight and dose over 3 minutes using an Ascor AP 22 syringe pump. The results were recorded within 3 minutes and drugs flushed with 0.5 ml normal saline. The BP was allowed to stabilize for 10 to 15 minutes, before further doses were infused. Six rats were used for each set of experiments.

**Experimental protocol**

Time zero (0) to twenty (20) minutes, the SHR was collected from the animal room and anaesthetized. While waiting for the anesthesia to take effect, all equipments were checked to see if they are properly set up. Catheters were heparinised. Tracheal tube was inserted into the trachea and the cannulation of the jugular vein and femoral artery done. Subsequently, the recording of the experimental parameters began.

Time twenty one (21) to forty (40) minutes, the animal was allowed to recover from the surgical procedure. Drugs and/or extract to be administered were diluted.

Time forty one (41) minutes, the animal was stable and the first dose infused.

Time forty five (45) to sixty (60) minutes, flushing was done, followed by recovery of the animal from drug effect, until initial base line was reached. Subsequent doses were then infused using the same procedure as that of the first dose.

**Experimental conditions of the animals**

To ascertain that the haemodynamic, respiratory and metabolic conditions of the rats were stable during the course of experiments, initial recordings of the cardiovascular parameters (SBP, DBP, MAP and HR) to be assessed during the study were done in a separate group of control SHRs over 3 hours, time much longer than the one needed for each experiment. During that period, no drug or plant extract was administered to the animal. Furthermore, in randomly selected rats, arterial blood sample (10 μl) was taken at the beginning (just after surgical preparation of the animal), and at the end of each experiment. Samples obtained were immediately sent in ice pack to PathCare Laboratory (Vet laboratory, Bellville, SA) for arterial blood gaz tests.

**Statistical analysis**

The results obtained are presented as mean values (± SEM). Statistical significance between means was calculated using the Student’s t-test and *p* value <0.05 was considered significant.

**Ethics considerations**

The study was approved by the ethics committee of the UWC (Ethics approval reference number 09/9/5) and was conducted according to the UWC rules and regulations in terms of animal experiments; and the European Community guidelines (EEC Directive of 1986; 86/609/EEC).

![Figure 1](http://www.biomedcentral.com/1472-6882/13/283)

**Figure 1** Effect of *A. gangetica* on BP (a) and HR (b). Values are presented as mean ± SEM. * indicates statistical significance.
Results

Experimental conditions

No statistically significant difference was observed in the SBP, DBP, MAP, HR, respiratory rate and arterial blood gaz test results during 3 hours the experiments were conducted.

Effect of A.gangetica on BP and HR in SHR

A. gangetica (10–400 mg/kg) significantly (<0.01), and dose-dependently decreased the maximum BP values obtained when compared to their respective values at baseline. A. gangetica also produced significant (<0.05) reductions in HR which were not dose-dependent (Figure 1).

Effect of angiotensin I on BP and HR in SHR

ANG I (3.1–100 μg/kg) significantly (<0.01), and dose-dependently increased the maximum BP values obtained when compared to their respective values at baseline. ANG I only produced significant (<0.05) change in HR at the 3rd and 2nd highest doses (25 and 50 μg/kg respectively, Figure 2).

Effect of angiotensin I co-infused with A.gangetica on BP and HR in SHR

The effects of ANG I (3.1 - 100 μg/kg) on the SBP, DBP, and MAP was significantly (p<0.001) inhibited by co-infusion with A. gangentica (200 mg/kg). The effect of ANG I on the HR was also significantly inhibited by A.gangetica (Figure 3).

Effect of angiotensin II on BP and HR in SHR

ANG II (3.1 – 50.0 μg/kg) significantly increased the SBP, DBP, and MAP in a dose-dependent fashion (Figure 4).

Figure 2 Effect of angiotensin I on BP (a) and HR (b). Values are presented as mean ± SEM. * indicates statistical significance.

Figure 3 Effect of ANG I co-infused with A. gangetica on the SBP (a), DBP (b), MAP (c), and HR (d). Values are presented as mean ± SEM. * indicates statistical significance.
Effect of angiotensin II co-administered with A. gangetica on the BP and HR in SHR
As shown in Figure 5, co-administration of A. gangetica (200 mg/kg) and ANG II (3.1 – 50.0 μg/kg), significantly (p<0.01) decreased the hypertensive effect of ANG II on the SBP, MAP and DBP, as well as its tachycardic effect.

Discussion
Effect of Asystasia gangetica on the blood pressure and heart rate
The results obtained in this study demonstrate that the ALE of A. gangetica decreases the BP in a dose dependent manner in SHR. The hypotensive effect observed (Figure 1) is concordant with the previous findings of Ramesar et al. [12] that both aqueous and methanol extracts of A. gangetica exhibited ACE inhibitory activity of 20% and 51% respectively in-vitro.

Effect of angiotensin II on the blood pressure and heart rate
The increase in BP is due to the direct vasoconstrictory actions of ANG II produced from ANG I in the presence of ACE in-vivo [20,21]. Meanwhile, the increase in HR is due to significant potentiation of the sympathetic activity, as well as a direct excitatory action in the heart produced by high levels of ANG II [18,22-27]. The decrease in the effect of ANG I on the HR at the dose ≥ 100 μg/kg may simply be down to Ang II shifting the operating ‘set-point’ for the regulation of sympathetic outflow to a higher BP [24,28].

Is the hypotensive effect of A. gangetica mediated by the inhibition of ACE?
The significant reductions in SBP, DBP and MAP produced by co-infusing the ALE of A. gangetica with ANG...
Ang II can be formed at the higher doses.

ANG I (Figure 3) when compared to the BP values observed with infusing ANG I alone (Figure 2) can be attributed to A. gangetica inhibiting the conversion of ANG I into ANG II, a powerful vasoconstrictor [29-32]. The significant reductions in HR observed with the co-infusion of A. gangetica with increasing doses of ANG I (Figure 3), as opposed to the absence of change, or even significant increases observed at some doses with the infusion of ANG I alone (Figure 2), could be attributed to lesser quantities of ANG II being produced. Since the dose of A. gangetica is fixed, it is expected that less ANG II can be produced at the lower doses of ANG I, while more ANG II can be formed at the higher doses.

Are angiotensin II receptors involved in the mechanism of action of Asystasia gangetica?

ANG II significantly increased the BP (Figure 4), due to the activation of ANG II receptors (AT1) in the vascular smooth muscle cells, resulting in increased vasoconstriction, decreased renal blood flow and renal tubular sodium re-uptake, increased aldosterone and vasopressin secretion [30-36]. The co-administration of the ALE of A. gangetica with ANG II (Figure 5) significantly inhibited the effect of ANG II alone (Figure 4) on BP. This antihypertensive effect was associated with significant reductions in HR. This gives more credence to the opinion that A. gangetica contain chemical compounds, which act by blocking the actions of ANG II at its various receptors mentioned above.

Limitations of the study

The angiotensin II (AT1) receptor mediates all of the known physiological actions of ANG II in the cardiovascular, renal, neuronal, endocrine, hepatic and other target cells [29,33,37-39]. Therefore, it is important to investigate the specific action(s) of the crude ALE, and its constituents at each target cell, as well as, the involvement of the alpha-1, beta-1, and presynaptic alpha-2 adrenoceptors; cholinergic receptors and calcium channels in the mechanism of action of A. gangetica.

Conclusions

A. gangetica decreases the blood pressure and heart rate in SHR. This action might be secondary to inhibition of ACE and the ANG II receptors. It may also involve a direct inhibitory action on the heart muscle.

Abbreviations

A. gangetica: Asystasia gangetica; ACE: Angiotensin I converting enzyme; ALE: Aquous leaf extract; ANG I: Angiotensin I; ANG II: Angiotensin II; AT1: Angiotensin II type 1 receptor; BP: Blood pressure; Bpm: Beats per minute; DBP: Diastolic blood pressure; DMSO: Dimethylsulfoxide; HR: Heart rate; MAP: Mean arterial pressure; mHg: Millimetres of mercury; SBP: Systolic blood pressure; SEM: Standard error of mean; SHR: Spontaneously hypertensive rats; UWC: University of the Western Cape.

Competing interests

The authors declare that they have no competing interests.

Authors’ contributions

PM conceived the idea of the study, designed the study and participated in the acquisition, analysis, and interpretation of data. IR participated in the conceptualization of the design, acquisition, analysis, and interpretation of data, and carried out the technical aspect of the study. All authors read and approved the final manuscript.

Authors’ information

PM has a PhD in Pharmacology, MMed in Cardiology, MBChB in medicine and BSc in human biology. PM is currently Professor of Pharmacology in the School of Pharmacy, University of the Western Cape, South Africa. IR has a PhD in Pharmaceutical Sciences, MSc and BSc Honours degrees in Human Physiology. IR is currently a Senior Lecturer in Physiology, at the National University of Science and Technology, Bulawayo, Zimbabwe.

Acknowledgement

The authors will like to acknowledge the assistance of Jo-anna Amunjela, Deon Hanse, Grace Hikumwah, Faydz Hoedemaker, Mantso Motsoaole, Paul Mwandingi, Xolani Sambo, and Vinesh Jeavan in the extraction of the crude aqueous leaf extract of A. gangetica, the care of the SHR used, and during the in-vivo experiments.

Author details

1Discipline of Pharmacology, School of Pharmacy, University of the Western Cape, Private Bag X17, Bellville 7535, South Africa. 2Division of Basic Medical Sciences, Faculty of Medicine, National University of Science and Technology, PO Box AC 939, Ascot, Bulawayo, Zimbabwe.

Received: 8 July 2013 Accepted: 24 October 2013

Published: 26 October 2013

References

1. Gurt-Fakim A: Medicinal plants: Traditions of yesterday and drugs of tomorrow. Mol Aspects Med 2006, 27(1):1-93.
2. Samuelsson G: Drugs of Natural Origin: a Textbook of Pharmacognosy. Stockholm: Swedish Pharmaceutical Press; 2004.
3. Balick MJ, Cox PA: Plants, People, and Culture: the Science of Ethnobotany. New York: NY Scientific American Library; 1997.
4. Scriba GE, Sweetman SC: Martindale: The complete drug reference. Pharmacognosy. Stockholm: Swedish Pharmaceutical Press; 2004.
5. Volpe M, Tocci G: Rationale for triple fixed-dose combination therapy with an angiotensin II receptor blocker, a calcium channel blocker, and a thiazide diuretic. Vasc Health Risk Manag 2012, 8:371–383.
6. Tocci G, Paneni F, Passerini J, Volpe M: Triple combination therapy to improve blood pressure control: experience with olmesartan-amlopidine-hydrochlorothiazide therapy. Expert Opin Pharmacother 2012, 13(18):2687–2697.
7. Laurent S, Schlaich M, Esler M: New drugs, procedures, and devices for hypertension. Lancet 2012, 380(9841):591–600.
8. Carvalho MF, Romano-Lieber NS, Bergsten-Mendes G, Secoli SR, Ribeiro E, Lebrão ML, Duarte YA: Polypharmacy among the elderly in the city of Sao Paulo, Brazil - SABE Study, Rev Bras Epidemiol 2012, 15(4):817–827.
9. Dunn SP, Holmes DR Jr, Moliterno DJ: Drug–drug interactions in cardiovascular catheterizations and interventions, JACC Cardiovasc Interv 2012, 5(12):1195–1208.
10. Lee WW, Pang KK, Hui KC, Kwok JC, Leung SL, Yu DS, Lee DT: Medication adherence: Is it a hidden drug-related problem in hidden elderly? Geriatr Gerontol Int 2013, 13(4):978–985.
11. Adeyemi OO, Aigbe FR, Uyaiabasi NG: Analgesic and anti-inflammatory activities of the aqueous stem and leaf extract of Asystasia gangetica (Linn) T. Anderson. Hug Q J Hosp Med 2011, 21(2):129–134.
12. Ramasar S, Sajnath H, Govender T, Mackraj J: Angiotensin I-converting enzyme inhibitor activity of nutritive plants in KwaZulu-Natal. J Med Food 2008, 11(2):331–336.
13. Akah PA, Ezike AC, Nwafor SV, Okoli CO, Enwereem NM: Evaluation of the anti-asthmatic property of Asystasia gangetica leaf extracts. J Ethnopharmacol 2003, 89(1):25–36.
14. Kokwaro JO. Medicinal plants of east Africa. 3rd edition. University of Nairobi Press; 2009:478.

15. Sudhakar M, Rao CV, Rao PM, Raju DB, Venkateswarlu Y: Antimicrobial activity of Caesalpinia pulcherrima, Euphorbia hirta and Asystasia gangeticum. Fitoterapia. 2006, 77(3):378–380.

16. Suzuki A, Kagawa D, Ochiai R, Tokimitsu J, Saito J: Green coffee bean extract and its metabolites have a hypertensive effect in spontaneously hypertensive rats. Hypertension Research. Official Journal of the Japanese Society of Hypertension 2002, 25(1):99–107.

17. Mackraj I, Ramesar S, Singh M, Govender T, Bainjath H, Singh R, Gathiram P: The in vivo effects of Tulbahia violacea on blood pressure in a salt-sensitive rat model. J Ethnopharmacol 2008, 117(2):263–269.

18. Isaacson JS, Reid IA: Importance of endogenous angiotensin II in the cardiovascular responses to sympathetic stimulation in conscious rabbits. Circulation research 1990, 66(3):662–671.

19. Hearse DJ, Sutherland FJ: Experimental models for the study of cardiovascular function and disease. Pharmacol Rev 2000, 41(6):597–603.

20. Pende A, Dallegrin F: Renin-angiotensin antagonists: therapeutic effects beyond blood pressure control? Curr Pharm Des 2012, 18(7):1011–1020.

21. Bakris GL: Are There Effects of Renin–Angiotensin System Antagonists Beyond Blood Pressure Control? Am J Cardiol 2010, 105(Suppl 21A):29A.

22. Kumagai K, Reid IA: Angiotensin II exerts differential actions on renal nerve activity and heart rate. Hypertension 1994, 24(4):451–456.

23. Averill DB: Neurochemical and peptidergic pathways of the baroreflex arc in the medulla oblongata: an introduction. Brain Res Bull 2000, 51(2):103–105.

24. Averill DB, Diz DI: Angiotensin peptides and baroreflex control of sympathetic outflow: pathways and mechanisms of the medulla oblongata. Brain Res Bull 2000, 51(2):119–128.

25. Diz DI, Garcia-Espinosa MA, Gallagher PE, Ganten D, Ferrario CM, Averill DB: Effects of aqueous leaf extract of Tulbahia violacea on blood pressure in a salt-sensitive rat model. J Ethnopharmacol 2008, 117(2):263–269.

26. Marchesi C, Paradis P, Schiffrin EL: Interaction of endothelial nitric oxide and angiotensin in the circulation. Pharmacol Rev 2007, 59(1):54–87.

27. Skrbic R, Igic R: Renin-angiotensin system dysfunction in hypertension: recent advances and perspectives. Br J Pharmacol 2003, 139(2):191–202.

28. Toda N, Ayajiki K, Okamura T: Role of the renin-angiotensin system in vascular inflammation. Trends Pharmacol Sci 2008, 29(7):367–374.

29. Ferrario CM: Role of angiotensin II in cardiovascular disease therapeutic implications of more than a century of research. Journal of the Renin-Angiotensin-Aldosterone System: JRAAS 2006, 7(1):3–14.

30. Unger T: Stoppelmaer R: Rationale for Double Renin-Angiotensin-Aldosterone System Blockade. Am J Cardiol 2007, 100(3, Supplement 1): S2–S31.

31. Kobori H, Nangaku M, Navar LG, Nishiyama A: The intrarenal renin-angiotensin system: from physiology to the pathobiology of hypertension and kidney disease. Pharmacol Rev 2007, 59(3):251–287.

32. Shimizu F, Kasai T, Takamata A: Involvement of central angiotensin II type 1 receptors in LPS-induced systemic vasopressin release and blood pressure regulation in rats. J Appl Physiol(Bethesda, Md: 1985) 2009, 106(6):1943–1948.

33. Siragy HM: Angiotensin II compartmentalization within the kidney: effects of salt diet and blood pressure alterations. Curr Opin Nephrol Hypertens 2006, 15(1):50–53.

34. Navar LG, Harrison-Bernard LM, Imig JD, Wang CT, Cervenka L, Mitchell KD: Intrarenal angiotensin II generation and renal effects of AT1 receptor blockade. Journal of the American Society of Nephrology: JASN 1999, 10(Suppl 12):S266–S272.

35. Head GA: Role of AT1 receptors in the central control of sympathetic vasomotor function. Clin Exp Pharmacol Physiol Suppl 1996, 3:593–598.

Cite this article as: Mugabo and Raji: Effects of aqueous leaf extract of Asystasia gangetica on the blood pressure and heart rate in male spontaneously hypertensive Wistar rats. BMC Complementary and Alternative Medicine 2013 13:283.

Submit your next manuscript to BioMed Central and take full advantage of:

• Convenient online submission
• Thorough peer review
• No space constraints or color figure charges
• Immediate publication on acceptance
• Inclusion in PubMed, CAS, Scopus and Google Scholar
• Research which is freely available for redistribution

Submit your manuscript at www.biomedcentral.com/submit