EVIDENCE OF A FUNCTIONAL RENIN-ANGIOTENSIN SYSTEM IN THE CHANNEL CATFISH (*ICTALURUS PUNCTATUS*)

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Abstract.–Salination of freshwater (FW) bodies has the potential to affect homeostatic regulation of osmotic and volume balance in FW organisms. The renin-angiotensin system (RAS) plays an important role in volume balance by maintaining blood pressure in marine and seawater acclimated euryhaline fish, but little is known about the RAS in FW adapted fish. The purpose of the present study was to first determine if the FW channel catfish (*Ictalurus punctatus*), demonstrates evidence of a functional RAS. Channel catfish (n = 6) were implanted with a catheter in the dorsal aorta to measure dorsal aortic pressure (P_{DA}) and infuse drugs. Infusion of [Asn^1,Val^5,Asn^9]-angiotensin I (ANGI) at 100, 400, and 1000 ng/kg significantly increased P_{DA} in a dose dependent manner ($P < 0.05$). Pretreatment with 2 mg/kg of the angiotensin converting enzyme inhibitor, Captopril (CAP), essentially eliminated the pressor response to the highest dose of ANGI ($P < 0.05$). Finally, infusion of 400 ng/kg [Asn^1,Val^5]-angiotensin II (ANGII) significantly increased P_{DA} from baseline ($P < 0.05$). The results suggest that channel catfish appear to have an operational RAS and may serve as a suitable model in which to study the role of ANGII in blood pressure regulation in FW adapted fish.

Keywords: renin-angiotensin, channel catfish, *Ictalurus punctatus*, osmoregulation

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Bodies of freshwater (FW) potentially face a state of increased salinity due to the thermal expansion of seawater (SW) (Titus & Narayanan 1996), dispersion of irrigated saline soils (Ingersoll et al. 1992; Nielsen et al. 2003), runoff from winter road salting (Novotny et al. 2008), and hurricane storm surge (Steyer et al. 2005). For example, in a FW marsh zone of the Terrabonne Basin in south central Louisiana, the storm surge from Hurricane Rita produced a spike in salinity to 17 ppt and remained above 6 ppt when measured nearly 3 months following the hurricane (Steyer et al. 2005). Such increases in environmental salinity may produce fluid shifts in FW organisms affecting vascular volume and blood pressure. As a result, homeostatic balance, and possibly survival, of FW fish in the face of salinity change may depend on species-specific efficiency to regulate osmolarity and volume.

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The renin-angiotensin system (RAS) is a highly conserved system that plays a key role in the regulation of blood pressure during osmotic and volume challenges in most vertebrates. Phylogenetic analysis of genes of the major constituents of the RAS indicates that all of the major components of the RAS are present in bony fish (Fournier et al. 2012). These authors further noted that although lampreys appear to lack genes coding for angiotensinogen and renin, plasma angiotensin II (ANGII) doubles in response to volume depletion, indicating an early Paleozoic appearance of the important role of ANGII in fluid homeostasis. It is widely held that one important functional aspect of ANGII is to act as a dipsongenic factor during states of hypovolemia or cellular dehydration. A study in dogfish (*Squalus acanthias*), demonstrated that the angiotensin converting enzyme inhibitor, Captopril (CAP), greatly reduced basal drinking rates and blocked the dipsogenic response to administration of angiotensin I (ANGI), while having no effect on a strong dipsogenic effect produced by ANGII infusion (Anderson et al. 2001). Earlier studies demonstrated a similar response in the SW acclimated, euryhaline flounder (*Platichthys flesus*) (Carrick & Balment 1983). These authors later showed that infusion of ANGII into FW acclimated flounder stimulated drinking and increased blood pressure (Balment & Carrick 1985). In the euryhaline bull shark (*Carchathinus leucas*), transfer from FW to 75% SW produced significant increases in plasma ANGII within 12 hours (Andersen et al. 2006). In contrast, the fall in arterial blood pressure, drinking rate, and plasma ANGII that accompanied infusion of CAP into SW acclimated eels (*Anguilla japonica*), was followed by a rise in plasma ANGII after termination of CAP infusion, but without a concomitant rise in drinking or blood pressure (Takei & Tsuchida 2000). This study suggests that CAP has longer term, nonspecific effects on other systems that may affect volume balance and calls into question earlier conclusions supporting a strong role of ANGII as a dipsongenic factor. If true, the principal effect of ANGII may well be the more rapid, direct or indirect actions that affect vascular resistance and cardiac function.

Infusion of ANGII has been shown to acutely increase blood pressure in a number of fish. Systemic infusion of ANGII acutely increases blood pressure in the Atlantic hagfish (*Myxine glutinosa*)

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(Carroll & Opdyke 1982), spiny dogfish (*Squalus acanthias*) (Carroll & Opdyke 1982), trout (*Oncorhynchus mykiss*) (Lancien et al. 2012), oyster toadfish (*Osanus tau*) (Nishimura & Bailey 1982; Qin et al. 1999), and American eel (*Anguilla rostrata*) (Nishimura et al. 1978; Oudit & Butler 1995; Bernier et al. 1999). With the exception of the Holostean bowfin (*Amia calva;* cf. Butler et al. 1995), it appears that most studies in fish that have determined the acute effects of systemically administered ANGII on blood pressure have been performed in marine adapted or euryhaline fish. Little data is available regarding the effect of ANGII on blood pressure in teleosts adapted to FW. Widely distributed throughout many FW systems of North America, the channel catfish (*Ictalurus punctatus*) can also be found in brackish coastal environments (Perry 1969). The upper limit of salinity tolerance has been determined for these fish to be 11 ppt (Perry & Avault 1969), or about one-third that of SW. Although channel catfish have limited salinity tolerance, these studies indicate this species to be a reasonable FW model in which to study ANGII.

A better understanding of the physiological responses of FW adapted species to changes in water salinity will allow improved predictive models of how factors such as thermal expansion of seawater (SW), dispersion of irrigated saline soils, runoff from winter road salting, and hurricane storm surge will impact FW ecosystems. As a first step, the purpose of the present study was to test the hypothesis that channel catfish have a functional RAS. To this end, the present study sought answers to the following questions: Does ANGI increase blood pressure in the channel catfish? If so, does administration of an angiotensin converting enzyme (ACE) inhibitor diminish the effect of ANGI to increase blood pressure? Does direct infusion of ANGII increase blood pressure?

MATERIALS & METHODS

Experimental animals.–Channel catfish (n = 6; 365.2 g ± 28.6 SE), acquired from a local fish supplier, were transported to Tarleton State University and housed in 400-L structural foam tanks filled with chlorine-free, filtered tap water (pH = 8.6 ± 0.1 SE) that was constantly

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aerated and recirculated through a biological filter. Animals were maintained at 20° C under a 14L:10D light cycle and provided commercially available food pellets 3 times per week. All housing and experimental procedures were approved by the Tarleton State University Institutional Animal Care and Use Committee (Animal Use Protocol #T-05-012-2015). Animals were allowed to acclimate to the laboratory conditions for at least one wk prior to any experimental procedures.

Chemicals and drugs.–Heparinized Cortland's solution was prepared by adding 1000 USP units/mL of heparin sodium to sterilized Cortland's saline for FW fish. This solution was used to maintain patency of catheters and as the vehicle for intravascular drug infusions. Treatment drug [Asn^1,Val^5, Asn^9]-Angiotensin I (ANGI) (Sigma-Aldrich; St. Louis, Missouri) was diluted in sterile, deionized water and frozen as 0.3-mL aliquots of 500 ng/mL, 2000 ng/mL, and 5000 ng/mL. Treatment drug [Asn^1, Val^5]-Angiotensin II (ANGII) (Sigma-Aldrich; St. Louis, Missouri) was diluted in sterile, deionized water and frozen as 0.3-mL aliquots of 2000 ng/mL. Frozen aliquots of ANGI and ANGII were later thawed and infused as a volume that produced the correct dose based on body weight. The volumes of ANGI or ANGII prepared for infusion ranged from 0.08 to 0.12 mL. Captopril (CAP) (Sigma-Aldrich; St. Louis, Missouri) was prepared as needed by dissolving in heparinized Cortland's solution. Additionally, Ethyl-3-aminobenzoate methanesulfonate (MS-222) (Sigma-Aldrich; St. Louis, Missouri) was utilized as the anesthetic for surgical procedures.

Dorsal aorta catheter implantation.–Channel catfish were anesthetized by placement in 20 L of water from the original holding tank containing 0.1 g/L MS-222. Fish were moved to a surgical table and artificially ventilated by circulating aerated, anesthetic solution through the gills at 1.5 L/min. A dorsal aorta (DA) catheter was implanted following procedures previously described by Burleson and Smatresk (1990). Briefly, tapered polyethylene tubing (PE-50; BD Intramedic®; Franklin Lakes, New Jersey) was inserted into the DA via a small, midline incision in the dorsal buccal cavity. The catheter tubing was then secured by two sutures to the roof of the buccal cavity and

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exteriorized through a snout tube. The catheter was filled with heparinized Cortland's solution and plugged for later access. Following the surgical procedure, fish recovered for at least 48 hours in a 40-L recovery tank filled with continuously filtered and aerated water obtained from the original holding tank. Prophylactic treatment with tetracycline powder (T.C. Tetracycline, Mars Fishcare North America; Chalfont, Pennsylvania) was mixed into the recovery tank at a dose of 12.5 mg/L. Sections of PVC pipe measuring 45 cm long x 7.5 cm diameter were placed in the tank to provide cover for the fish. After recovery, fish were transferred in the PVC cover tube to a rectangular experimental chamber measuring 50 x 10 x 10 cm. Filtered, aerated water from a 100-L tank was circulated through the experimental chamber at 1.5 L/min.

Measurement of hemodynamic parameters.–Pulsatile arterial pressure signals were monitored via the DA catheter using a Cobe CDX III pressure transducer (Argon; Athens, Texas). Calibrated signals from the transducer were amplified using an ETH-400 bridge amplifier (CB Sciences; Australia), digitized using a PowerLab 4sp (ADInstruments; Australia), and visualized and recorded on a personal computer using LabChart software v. 7.4 (ADInstuments; Australia). Mean DA pressure (P_{DA}) and heart rate (HR) were calculated from the raw pulsatile signal using software protocols in LabChart and visualized on separate channels.

Experimental protocol.–Once the hemodynamic monitoring of fish commenced, fish were allowed 1 to 2 hrs to establish baseline P_{DA} and HR before experimentation. ANGI doses of 100, 400, and 1000 ng/kg were administered as 0.2-mL bolus infusions into the DA. Each dose was flushed with 0.2 mL of Cortland's heparinized saline, and 30 to 45 minutes separated each dose to allow reestablishment of baseline hemodynamic parameters. The peak change of P_{DA} and associated HR to each dose of ANGI was recorded as a 10-sec average around the highest observed P_{DA} signal in response to the ANGI dose, minus a one-min average of P_{DA} and HR just prior to infusion of the ANGI dose. After recovery from the final 1000-ng/kg dose of ANGI, 2 mg/kg CAP was infused into the DA as a slow, bolus infusion and flushed with 0.2

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mL Cortland's heparinized saline. After 30 min, a one-min average of P_{DA} and HR was compared to the average baseline P_{DA} and HR, respectively, measured one min prior to CAP infusion, and the change in P_{DA} and HR in response to ANGI 1000 ng/kg was repeated. Finally, the P_{DA} and HR response to ANGII was determined. After recovery from the post-CAP ANGI infusion, ANGII 400 ng/kg was infused into the DA as a 0.2-mL bolus infusion and flushed with 0.2 mL Cortland's heparinized saline. A 10-sec average around the peak P_{DA} and its associated HR response to ANGII was recorded. Baseline P_{DA} and HR were recorded as the average P_{DA} and HR during the last minute prior to ANGII infusion.

Statistical analysis.–The effects of ANGI doses on the change in P_{DA} and HR from Baseline were determined on a personal computer with SPSS software (IBM® SPSS Statistics®; IBM Corporation; Armonk, New York) using one-way ANOVA with repeated measure. Significant differences ($P < 0.05$) as determined by ANOVA were further evaluated using a False Discovery Rate procedure. The mean P_{DA} and HR before and 30 min following intra-arterial infusion of CAP was analyzed via paired sample *t*-test. Finally, a paired sample *t*-test was used to compare baseline P_{DA} and HR to the peak response of P_{DA} and HR, respectively, to intra-arterial infusion of ANGII. Statistical significance for paired sample *t*-tests was $\alpha = 0.05$. All results are reported as mean ± SE.

RESULTS

Infusion of ANGI at doses of 100, 400, and 1000 ng/kg produced a dose dependent increase in mean P_{DA} over baseline by 2.6 ± 0.9 mmHg, 6.6 ± 1.1 mmHg, and 12.8 ± 1.1 mmHg, respectively (Fig. 1). Each dose produced an effect that was statistically significant compared to the previous dose ($P < 0.05$) (Fig. 1). Pretreatment with the ACE inhibitor, CAP, resulted in a P_{DA} response to 1000 ng/kg ANGI that was significantly different from the 400 and 1000 ng/kg ANGI administered prior to CAP ($P < 0.05$), and effectively reduced the pressor response

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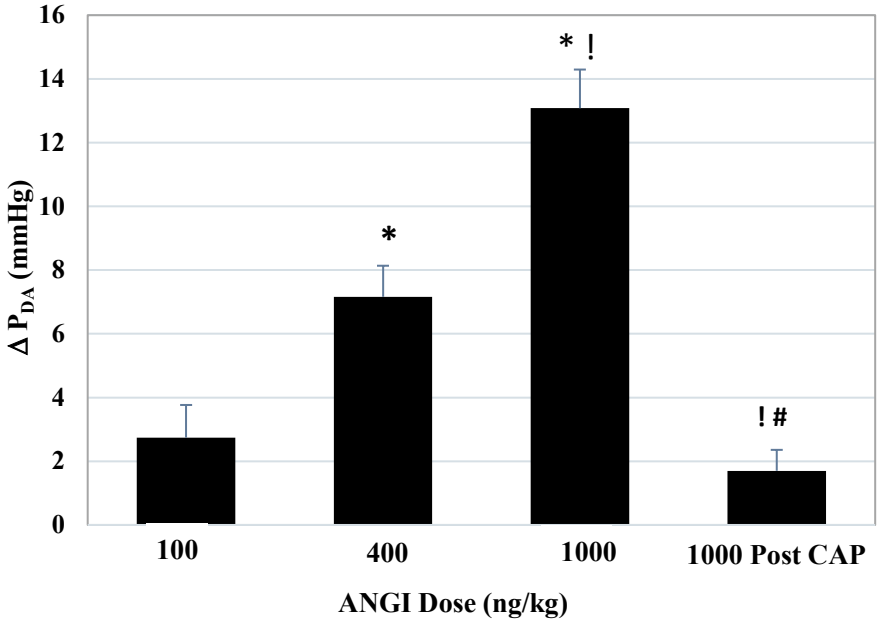

Figure 1. Dose effect of Angiotensin I on dorsal aortic pressure. Peak change in dorsal aortic pressure from baseline (ΔP_{DA}) in response to bolus, dorsal aorta infusions of angiotensin I (ANGI) at doses of 100, 400, and 1000 ng/kg, and the ΔP_{DA} to 1000 ng/kg ANGI 30 min following infusion of Captopril (CAP). Error bars indicate SE. (* $P < 0.05$ compared with 100; ! $P < 0.05$ compared with 400; # $P < 0.05$ compared with 1000).

to a level not different than that observed in response to the 100 ng/kg dose.

The ACE inhibitor, CAP, affected baseline blood pressure (Fig. 2). At rest, baseline P_{DA} averaged 20.1 ± 1.2 mmHg. Thirty min following infusion of CAP, mean P_{DA} was measured to be 15.7 ± 1.6 mmHg, which was significantly below the baseline value ($P < 0.05$) (Fig. 2).

Infusing 400 ng/kg ANGII affected baseline P_{DA} (Fig. 3). Note that baseline P_{DA} prior to ANGII infusion followed the administration of CAP. Bolus infusion of 400 ng/kg ANGII increased mean P_{DA} to 27.1 ± 2.7 mmHg from a baseline of 14.7 ± 2.5 mmHg ($P < 0.05$) (Fig. 3).

The response of HR to the experimental procedures was variable. The change in HR in response to 100, 400, and 1000 ng/kg ANGI and

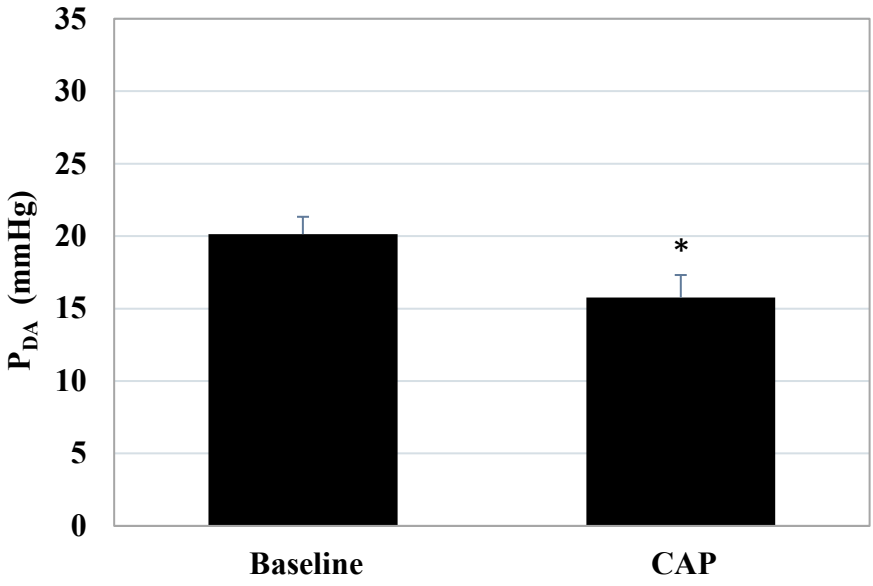

Figure 2. Effect of Captopril on Baseline dorsal aortic pressure. Dorsal aortic pressure (P_{DA}) prior to (Baseline) and 30 min following bolus, dorsal aorta infusion of 2 mg/kg Captopril (CAP). Error bars indicate SE. (* $P < 0.05$ compared to Baseline).

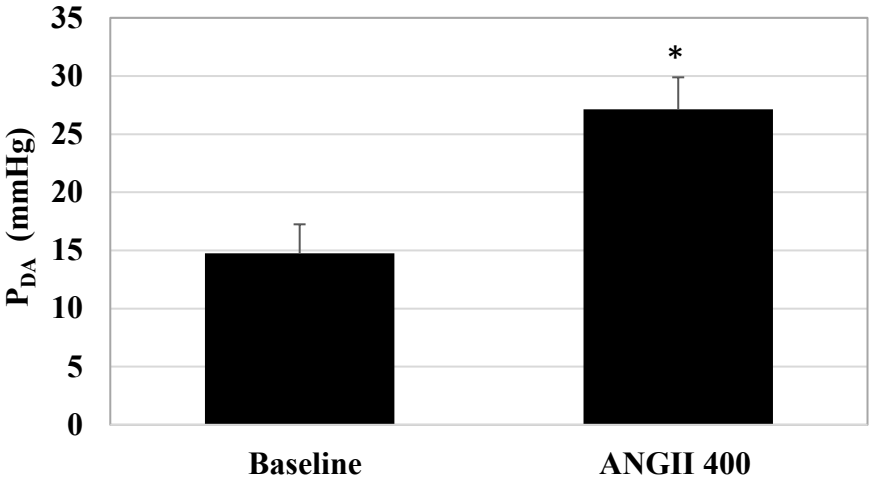

Figure 3. Effect of Angiotensin II on dorsal aortic pressure. Dorsal aortic pressure (P_{DA}) prior to infusion of angiotensin II (Baseline) and the peak response of P_{DA} to bolus, dorsal aorta infusion of 400 ng/kg of angiotensin II (ANGII 400). Error bars indicate SE. (* $P < 0.05$ compared to Baseline).

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the HR response to ANGI 1000 ng/kg following 2mg/kg CAP was -2.0 ± 2.5, 3.2 ± 3.1, 2.7 ± 4.0, and -4.9 ± 2.8 bpm, respectively, and was not statistically significant (Fig. 4). The HR measured 30 min after CAP (41.1 ± 3.9 bpm) did not change from Baseline (37.3 ± 2.4 bpm) (Fig. 5). Infusing 400 ng/kg ANGII resulted in a HR of 43.7 ± 3.9 bpm, which was not significant compared to Baseline (37.3 ± 4.5 bpm) (Fig. 6).

DISCUSSION & CONCLUSIONS

The primary objective of the present study was to determine if the FW channel catfish, *Ictalurus punctatus*, shows evidence of a functional RAS. The key observations were that: 1) ANGI produced a dose-dependent increase in P_{DA} that could be effectively eliminated by pretreatment with an ACE inhibitor, and 2) that direct infusion of ANGII produced a rapid and robust increase mean P_{DA}. These observations lead to the general conclusion that channel catfish possess the key operational components of the RAS, namely an operational converting enzyme and an acute pressor response to ANGII.

The present study demonstrated a dose-dependent increase in P_{DA} in response to bolus infusions of ANGI. This is consistent with previous observations in the dogfish (Opdyke & Holcombe 1976), toadfish (Qin et al. 1999), and American eel (Nishimura et al. 1978). Furthermore, pretreatment with the ACE inhibitor, CAP, eliminated the pressor effect of ANGI in the present study. Previous studies in the eel noted that ACE inhibition, while also inhibiting the pressor response to ANGI, decreased baseline blood pressure and increased plasma renin activity (Nishimura et al. 1978). This observation led the authors to suggest that ANGII might importantly contribute to the baseline, or tonic, level of blood pressure regulation. In the present study, CAP significantly decreased resting P_{DA} by nearly 25%. It is tempting to conclude that ANGII (synthesized from ANGI) might significantly contribute to the tonic, resting P_{DA}, however, further research is necessary to reach this conclusion. A study in eels demonstrated a dose-dependent fall in arterial blood pressure, inhibition of drinking, and a decrease in plasma ANGII in response to infusion of an ACE inhibitor. It is interesting to

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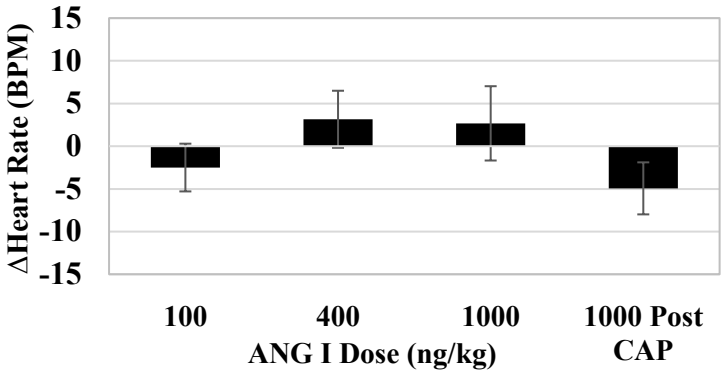

Figure 4. Dose effect of Angiotensin I on heart rate. Peak change in heart rate from baseline (ΔHR) in response to bolus, dorsal aorta infusions of angiotensin I (ANGI) at doses of 100, 400, and 1000 ng/kg, and 1000 ng/kg ANGI 30 min following infusion of Captopril (CAP). Error bars indicate SE.

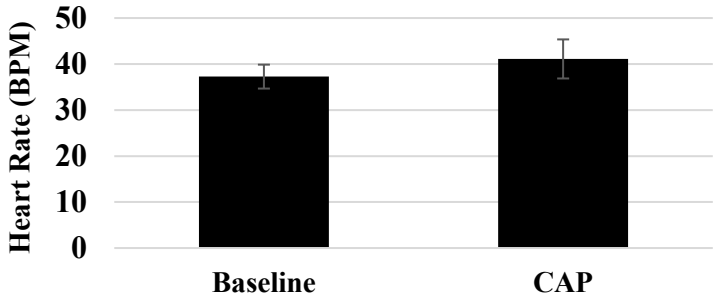

Figure 5. Effect of Captopril on Baseline heart rate. Heart rate (HR) prior to (Baseline) and 30 min following bolus, dorsal aorta infusion of 2 mg/kg Captopril (CAP). Error bars indicate SE.

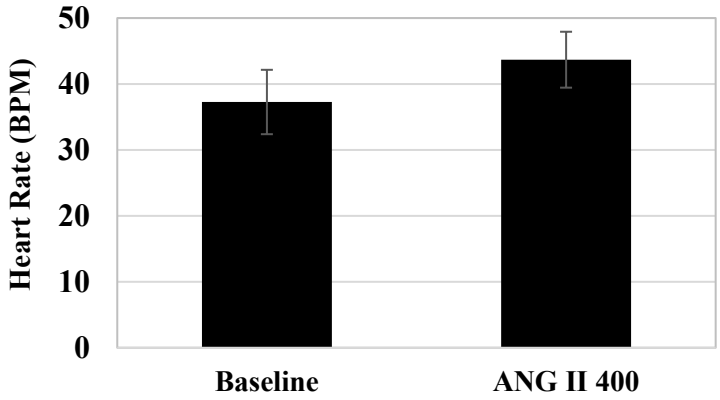

Figure 6. Effect of Angiotensin II on heart rate. Heart rate (HR) prior to infusion of angiotensin II (Baseline) and the peak response of HR to bolus, dorsal aorta infusion of 400 ng/kg of angiotensin II (ANGII 400). Error bars indicate SE.

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note that after the infusion of the ACE inhibitor was terminated, plasma ANGII levels returned toward pre-infusion levels, but both drinking behavior and arterial blood pressure remained suppressed (Takei & Tsuchida 2000). These data suggest that the effect of ACE inhibition to lower resting blood pressure and inhibit drinking involves mechanisms other than the RAS. Because ACE inhibitors are nonspecific carboxyl dipeptidase inhibitors that affect a number of proteases, it is possible that mechanisms involving other proteases are influencing the drinking behavior and blood pressure (Takei & Tsuchida 2000).

Previous studies have demonstrated a pressor response to ANGII infusion in both marine and euryhaline fish. Infusion of approximately 2000 ng/kg ANGII into the hagfish and dogfish produced changes in P_{DA} averaging 3.4 and 5 mmHg, respectively (Carroll & Opdyke 1982). In trout, infusion of 300 and 600 ng/kg ANGII increase P_{DA} by approximately 11 and 18 mmHg, respectively (Lancien et al. 2012). In the toadfish, P_{DA} increased by 12 mmHg following infusion of ANGII at 400 ng/kg (Qin et al. 1999) and infusion of 150 ng/kg ANGII increased P_{DA} by nearly 20 mmHg in the American eel (Oudit & Butler 1995). The more pronounced pressor responses observed in the trout, toadfish, and eel suggest that teleosts may be more sensitive to the pressor actions of ANGII than are cyclostomes and elasmobranches. The present study observed an increase in P_{DA} of 12.8 mmHg in response to infusion of 400 ng/kg ANGII. This response is consistent with responses observed in other teleost fish and suggests that the FW adapted channel catfish has a pressor sensitivity to ANGII that is similar to that observed in marine and euryhaline teleost fish.

Infusion of ANGI or ANGII had little effect on HR. Given the dose dependent increase in blood pressure in response to ANGI, it is notable that a baroreflex-mediated decrease in HR was not observed. Similar observations have been recorded in mammals. In rabbits, systemic ANGII acts reset arterial baroreflex control of HR to higher pressures through mechanisms involving the Area Postrema, a brain stem region located near the base of the fourth ventricle (Bishop & Sanderford 2000; Sanderford & Bishop 2002). The central resetting acts to blunt baroreflex suppression of HR when blood pressure is elevated with

ANGII. Interestingly, lesion of the Area Postrema in eels diminishes the dipsogenic action of ANGII (Nobota & Takei 2011), and intracerebroventricular infusion of ANGII into the fourth ventricle of trout at doses that have no systemic effect acts to increase HR and arterial blood pressure (LeMevel et al. 1994). Together these data provide evidence that central neural actions of ANGII might be an early, important hallmark of the RAS that is retained in mammals. Additional studies are needed to determine if the observed effects of ANGI and ANGII on HR in the present study are the result of central neural actions of ANGII to blunt arterial baroreflex suppression of HR.

In summary, the channel catfish appears to possess a functional RAS. A key question relates to what physiological stimuli lead to activation of the RAS and what role does ANGII play in this FW adapted teleost. Environmental and/or climate induced changes in FW salinity have the potential to push FW adapted organisms to the limits of their homeostatic acclimatization range. Because the channel catfish is widely abundant, easy to culture, and appears to have a pressor sensitivity to ANGII that is similar to marine and euryhaline teleost fishes, it is concluded that the channel catfish might be a suitable model organism for the study of ANGII-mediated mechanisms involved in the regulation of blood pressure of FW adapted fish.

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