insight into the oxidative processes related to the maintenance energetics and growth dynamics of this extraordinary fish.

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Vascular reactivity in hypertension: Altered effect of ouabain[1]

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Summary. Ouabain inhibits the relaxing effect of Ca^{2+} (but not of Mn^{2+}) on contractile responses in tail artery strips isolated from spontaneously hypertensive and normotensive rats. The magnitude of ouabain inhibition was greater in vascular strips from hypertensive rats suggesting a significant difference in basic membrane function in hypertensive vascular smooth muscle.

The cause of the elevated arterial pressure in most forms of hypertension is an increase in vascular resistance. The mechanism responsible for this increase in resistance is not well defined. It could be a structural increase in wall thickness of the resistance vessels or a functional increase in contraction of the smooth muscle of these vessels. The latter could be caused by an increase in neurogenic or circulating humoral vasoconstrictor activity or by a change in the vascular smooth muscle itself which makes the muscle more sensitive to any constrictor stimulation. The present study gives insight into the mechanism responsible for a difference that occurs in vascular smooth muscle of spontaneously hypertensive rats (SHR).

An increase in extracellular Ca^{2+} concentration of vascular smooth muscle has been shown to have a triphasic effect on the contractile response[2]: a) small increases in free Ca^{2+} above physiological levels (1.6 mM) produce an augmentation of contraction; b) larger increases in Ca^{2+} depress the

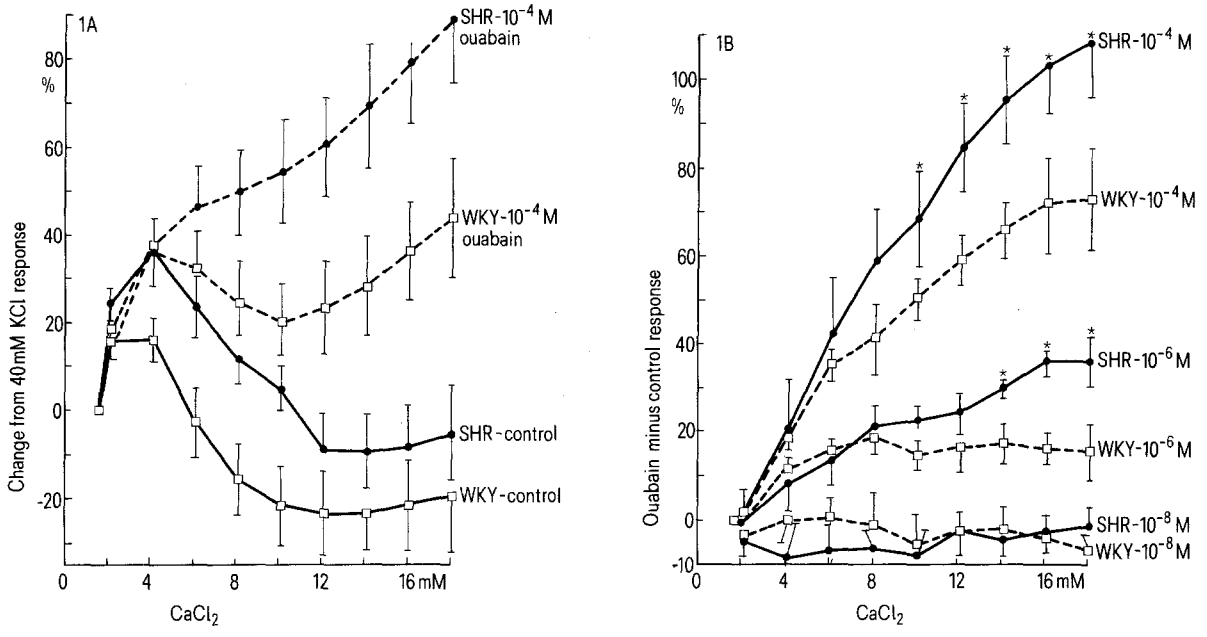

Fig. 1. *A* Effect of Ca^{2+} concentration on KCl contraction of tail artery strips from SHR and WKY. Helical strips of tail arteries from SHR and WKY were made to contract in response to 40 mM KCl in the presence and absence of ouabain. The bath concentration of $CaCl_2$ was then increased to the levels indicated. Changes in contractile tension are expressed as percent change from the 40 mM KCl response with 1.6 mM $CaCl_2$ in the bath. The mean contractile response to 40 mM KCl in each of the 4 groups before the addition of extra Ca^{2+} was: 1. SHR-control = 54 ± 6 mg; 2. WKY-control = 61 ± 8 mg; 3. SHR-10^{-4} M ouabain = 111 ± 10 mg; and 4. WKY-10^{-4} M ouabain = 121 ± 11 mg. Values are the mean ± SEM for 8 SHR and 8 WKY (2 helical strips per rat). *B* Ouabain inhibition of Ca^{2+} relaxation including some data from A. The measure of ouabain inhibition shown on the ordinate was determined by subtracting the control responses (no ouabain) of each animal from their respective treated responses (ouabain present). Statistical analysis (Student's t-test) of the responses at each Ca^{2+} concentration indicate that SHR responses were significantly ($p < 0.05$, marked by asterisk) greater than WKY at ouabain concentrations of 10^{-6} and 10^{-4} M. Integration of experimental values to determine a mean area for each curve showed SHR responses were different from WKY at the $p < 0.05$ level when 10^{-4} M ouabain was added, and at the $p < 0.10$ level when 10^{-6} M ouabain was added. Values are the means ± SEM for 4 SHR and 4 WKY (2 strips per rat.).

magnitude of contractile response because the plasma membrane is stabilized; and c) still higher concentrations of Ca^{2+} may produce a secondary increase in contraction. Regardless of etiology of experimental hypertension (renal, genetic or steroidal), isolated vascular smooth muscle from the hypertensive animal relaxes less in response to high concentrations of Ca^{2+} than does that from the normotensive animal[2]. This difference suggests that the membranes of vascular smooth muscle from hypertensive animals are more labile, and therefore require a higher concentration of Ca^{2+} to effect an equivalent membrane stabilization[2]. This altered vascular response is unrelated to the strain of rat or to the influence of increased wall stress produced by high systemic arterial pressure[3].

Recently, we have shown that membrane stabilization by Ca^{2+} is dependent on the electrogenic pumping of Na^+ and K^+ by Na^+-K^+ adenosine triphosphatase[4]. The basis for this interpretation is that ouabain prevents Ca^{2+} relaxation of vascular smooth muscle under conditions in which it has no effect on Mn^{2+} relaxation. We are now reporting that relaxation of vascular smooth muscle in response to Ca^{2+} is inhibited to a greater degree by ouabain in SHR than in Wistar Kyoto normotensive rats (WKY). This observation reflects a difference between the vascular smooth muscle of these 2 strains of rats. The difference results in a greater contraction of the smooth muscle from the hypertensive animal.

Materials and methods. Helical strips cut from tail arteries of adult female SHR and WKY[5] were mounted in a muscle bath containing physiological salt solution [PSS, aerated with 95% O_2 and 5% CO_2, and maintained at 37 °C][4]. Before the start of experiments, the strips were allowed to equilibrate for 60–90 min in PSS. The effects of cumulative additions of Ca^{2+} or of Mn^{2+} on the contractile response of the strips to 40 mM KCl were studied by adding appropriate volumes of 1.0 M $CaCl_2$ or 0.5 M $MnCl_2$ to the bath during the plateau period of the KCl response. In order to eliminate possible interactions of norepinephrine released from nerve terminals by potassium, the experiments were carried out in the presence of 10^{-5} M phentolamine, an alpha-adrenergic blocking agent.

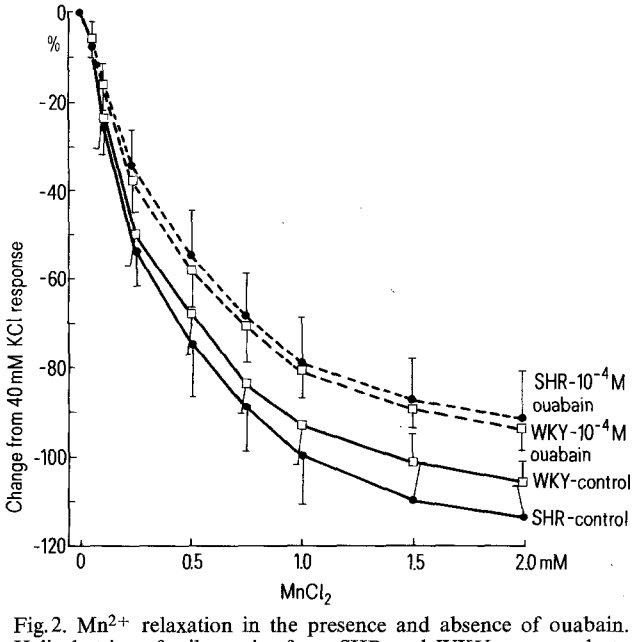

Fig. 2. Mn^{2+} relaxation in the presence and absence of ouabain. Helical strips of tail arteries from SHR and WKY were made to contract in response to 40 mM KCl in the presence and absence of ouabain (10^{-4} M). Mn^{2+} was then added to the bath to produce relaxation. Changes in contractile tension are expressed as percent change from the 40 mM KCl response. The mean contractile responses to 40 mM KCl in each of the 4 groups before addition of Mn^{2+} was: 1. SHR-control = 64 ± 10 mg; 2. WKY-control = 69 ± 9 mg; 3. SHR-10^{-} M ouabain = 122 ± 18 mg; and 4. WKY-10^{-4} M ouabain = 127 ± 10 mg. In control experiments on both SHR and WKY, the addition of Mn^{2+} at concentrations greater than 1.0 mM usually resulted in a depression of the contractile response which was below the baseline tension before the addition of 40 mM KCl. This caused the calculation of percent change to be greater than 100% and indicates that there is a component of resting tension in the unstimulated muscle which may be dependent upon the concentration gradient of Ca^{2+} across the plasma membrane. Values are the means ± SEM for 4 SHR and 4 WKY (2 strips per rat).

Results and discussion. Figure 1, A, depicts the effect of Ca^{2+} concentration on the KCl responses of tail artery strips from SHR and WKY in the presence and absence of 10^{-4} M ouabain. Increasing the Ca^{2+} concentration from 1.6 to 4.1 mM potentiated the contraction in response to KCl in all strips. The potentiation of contraction by this small increment in Ca^{2+} concentration was significantly greater in strips from SHR. Further elevation of the Ca^{2+} concentration resulted in a depression of the contraction in both SHR and WKY. Maximum relaxation was not as great in SHR as in WKY, and the concentration of Ca^{2+} required to produce an equivalent relaxation was greater in SHR.

Treatment of the strips with 10^{-4} M ouabain inhibited Ca^{2+} relaxation in both SHR and WKY. The magnitude of ouabain inhibition was determined by subtracting the control or untreated responses for each animal from their respective responses after treatment (ouabain present, figure 1, A). The effect of 3 different ouabain concentrations on this measure of ouabain inhibition (treated minus control response) is shown in figure 1, B. Ouabain at a concentration of 10^{-8} M did not significantly alter the effect of Ca^{2+} on KCl responses in either SHR or WKY. A small change from control responses occurred at 10^{-6} M ouabain in both SHR and WKY; and the change from control was significantly greater ($p < 0.05$) in strips from SHR than WKY at $CaCl_2$ concentrations greater than 12.1 mM. When 10^{-4} M ouabain was added to the bath, the strips from SHR responded to the addition of Ca^{2+} with a greater change from control than did strips from WKY. SHR responses were significantly greater than WKY at Ca^{2+} concentrations greater than 8.1 mM. In figure 1, A, it can be seen that 10^{-4} M ouabain completely prevents Ca^{2+} relaxation in vascular smooth muscle from SHR, whereas relaxation persists in that from WKY. From these observations we conclude that the magnitude of ouabain inhibition of Ca^{2+} relaxation in SHR is greater than that in WKY.

The greater inhibition of Ca^{2+} relaxation by ouabain in SHR than in WKY probably reflects a significant difference in membrane function in hypertensive vascular smooth muscle. Ouabain probably alters the contractile activity of smooth muscle via its specific inhibitory action on the Na^+-K^+ pump[6,7]. Thus, the present experiments suggest that the altered membrane function in hypertensive vascular smooth muscle may involve the electrogenic pumping of Na^+ and K^+. Alternatively, the number of Ca^{2+} channels in the plasma membrane of SHR or the regulation of intracellular Ca^{2+} by sarcoplasmic reticulum may differ in SHR and WKY[8,9]. These latter possibilities are probably not very important since relaxation of KCl responses by Mn^{2+} was not different in SHR and WKY in the presence or absence of ouabain (figure 2). Mn^{2+} produces relaxation of vascular smooth muscle by blockage of Ca^{2+} channels[10]. If a difference in the subcellular regulation of Ca^{2+} or the number of membrane channels for Ca^{2+} were important components of the ouabain inhibition, then it would have been expected that the difference

in SHR and WKY would also have been evident when Mn^{2+} was used as a relaxing agent.

These experiments on SHR confirm earlier observations made on SHR and also on renal and steroid-salt hypertension in rats[2,3]. They indicate that contraction of vascular smooth muscle from the hypertensive animal is less readily depressed by high concentrations of Ca^{2+} than is that from normotensive controls. The current studies provide evidence that the mechanism responsible for this intrinsic change in vascular smooth muscle in hypertension is an alteration in the electrogenic pumping of Na^+ and K^+ at the plasma membrane.

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Spruce budworm: Roles of pheromone components and analogues in male disruption and attraction[1]

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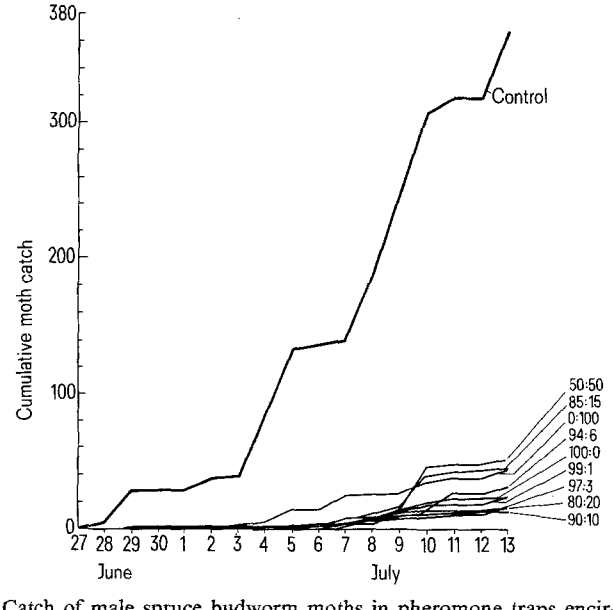
Summary. Male *Choristoneura fumiferana* are disrupted by atmospherically permeating any blend of the 2 sex pheromone components. The mechanisms of disruption and attraction are different and appear to result from decisions in the brain based on chemical and physical characteristics of the air and pheromone plumes surrounding a pheromone source.

At present there is an abundance of evidence that male moths, including the spruce budworm, are optimally attracted to only limited ratios of female sex pheromone components[3–5]. In the spruce budworm this range seems to be between 1 and 15% (Z)-11-tetradecenal (Z-TDA) in (E)-11-tetradecenal (E-TDA), with the best ratio about 3–4% (Z-TDA) in (E-TDA)[6].

In contrast to attraction, the pertinent question relating to disruption of male orientation to females by atmospheric permeation of pheromone is not what ratios of pheromone components will attract males, but rather, what ratios will prevent males from being attracted to a female. This distinction between attraction and disruption is rarely stressed and its significance more infrequently appreciated: there is no a priori reason to expect the optimally attractive ratios of pheromone components to be the only, or even the best, ratios for disruption. The objectives of this study were to determine what ratios of pheromone components would successfully disrupt orientation of the male eastern spruce budworm *Choristoneura fumiferana* (Clem.) and to analyze the effect of various pheromone analogues on orientation.

9 different isomeric ratios of the 2 sex pheromone components and 5 pheromone analogues, 11-tetradecen-1-ol acetate (TDA), 11-tetradecen-1-ol (TDOH), 11-epoxy-tetradecanal (epoxy-TDAL), 12-pentadecen-2-one (PDONE), and 11-dodecenal (DDAL), were synthesized. Each ratio or analogue was incorporated at a rate of 3% in 5 mm diameter and 10 cm long continuous slow release polyvinyl chloride plastic. These formulations were tested in 2 experimental locations 21 km apart near the village of St. Quentin, N.B. In each area 10 plots consisting of a center pheromone trap baited with synthetic 97:3 E:Z-TDA at an elevation of 1 m surrounded at a distance of 5 m by 8 equidistant identical test emitters were layed out 160–200 m apart. The 8 emitters released pheromone into the surrounding air at an estimated rate of 124 µg/h, or 8.0 mg/ha/h as measured by the method of Fitzgerald et al.[7]. The center trap in each plot of area I was surrounded by emitters containing 1 of the test pheromone ratios or containing controls made of polyvinyl chloride plastic formulation without pheromone. Area II was left untreated as a further control. The treatments of the areas were



Catch of male spruce budworm moths in pheromone traps encircled by 8 pheromone emitters. Each pheromone blend utilized is designated as a ratio of E-TDAL:Z-TDAL.