with 'related' reptile fossils, one could speculate that in the light of current knowledge of tuatara egg lipids and the corresponding slow lipid metabolic activities[14], these findings may help explain the survival of the tuatara over such a long period of time.

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Acetylcholine induced endothelial-dependent vasodilation increases as artery diameter decreases in the rabbit ear

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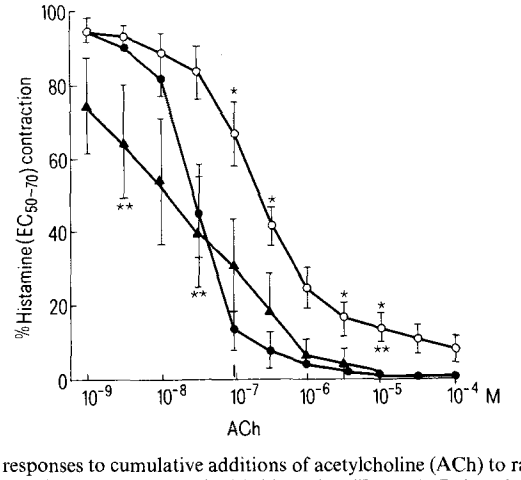
Summary. Isolated resistance vessels in the rabbit ear preconstricted with histamine were relaxed by acetylcholine by a proportionately greater amount than the central ear artery. The relaxation was antagonized by atropine and also by endothelium removal. Our studies represent the first direct evidence that endothelium-dependent dilation can occur in resistance vessels.
Key words. Acetylcholine; endothelium-dependent vasodilation; resistance vessel.

Furchgott and Zawadzki[1] discovered that the relaxation by ACh of isolated preparations of rabbit thoracic aorta and several other large and medium sized precontracted arteries is dependent on the presence of endothelial cells. Presumably because in vitro experiments using smaller vessels are technically difficult, no studies have appeared which analyze this phenomenon in resistance arteries. The aim of this study was to determine whether vasodilation of resistance vessels in the rabbit ear by ACh is endothelium-dependent and whether the dilation is quantitatively and qualitatively similar.

Male New Zealand White rabbits (2–3 kg) were stunned and exsanguinated. The central ear artery (CEA) (unstretched lumen diameter (ULD) ~ 300 μm), a main side branch (MSB) (ULD ~ 150 μm) of the CEA, and a terminal branch (TB) (ULD ~ 75 μm) of the MSB were removed and ring segments prepared. CEA segments were mounted on a standard tissue bath myograph[3]. The two smaller vessels were mounted on a smaller myograph using a modification[2] of a method used by Mulvany and Halpern[4]. Detailed procedures for isometric contraction and relaxation have been described in an earlier report[2].

Exogenous ACh caused a concentration-dependent relaxation of all arterial segments when they were initially constricted with histamine (H) (EC_{50}–EC_{70}) (fig.). Log IC_{50} values and the maximum relaxation produced by ACh at each concentration for the three vessels were compared by a one-way analysis of variance. Individual mean comparisons were performed using a multiple t-test with adjustment for multiple comparisons. $p < 0.05$ was accepted as a significant difference. By the criteria of IC_{50} value comparison, the TB was determined to be more sensitive to ACh than the CEA. The maximum relaxation produced by ACh did not differ significantly between the arteries (CEA, 92%; MSB, TB, 100%). The responsiveness to exogenous ACh was different in the three arteries as exhibited by significant differences in the concentration-effect curves (fig.).

The endothelium-dependency of the action of ACh was demonstrated in the CEA and MSB by the abolition of the relaxation response by mechanical removal of the endothelial layer (the arteries were rotated on their supporting wires). Rotated preparations of TB exhibited some relaxation of tone upon ACh addition – they relaxed 56–100% of initial relaxation. However,



Mean responses to cumulative additions of acetylcholine (ACh) to rabbit ear vessels initially constricted with histamine (EC_{50-70}). Relaxation responses are illustrated as percentage of the histamine contraction remaining upon acetylcholine addition in each experiment. Each point represents the mean ± SEM of different vascular segments (central ear artery (CEA) (○): n = 8; main side branch (MSB) (●): n = 4; terminal branch TB) (▲): n = 4). *Significant difference from MSB. **Significant difference from CEA.

at least a 2¼ log concentration increase of ACh was required to produce 50% of the second relaxation response. Rubbing of the tissue to remove the endothelium was accomplished in all arteries without significantly altering the contractile response to H. Incubation with atropine (10^{-5} M) blocked ACh-induced relaxation indicating that the response is mediated through muscarinic receptors. Explanations for the resistant portion of the vasodilation response to ACh after mechanical removal of the endothelial layer in the TB are that the tissues were 'inadequately' rubbed (still retained some endothelial cells) and/or the smooth muscle cells of the TB possess muscarinic receptors mediating vasodilation[5].

Our studies represent the first direct evidence that endothelium mediated dilation occurs in resistance vessels. It would seem reasonable that endothelial-based vasodilation should become greater as vessels become smaller, the internal elastic lamina thinner and more fenestrated and the intimal/media mass ratio and myoendothelial junction density greater[6]. Also the two smaller arteries studied possess intrinsic tone[2], and this would provide an appropriate background for dilation.

In conclusion, the determination that acetylcholine can cause dilation in vessels as small as 75 µm (ULD) and that this effect is proportionately greater in smaller vessels suggests that if there is a physiological role for endothelium-based relaxation, this would be more profound in smaller compared with larger arteries. The exact role of this mechanism awaits determination.

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Effects of gonadotropin-releasing hormone (LH-RH) on the pars distalis and testis of the Skipper frog, *Rana cyanophlyctis* (Schn.)[1]

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Summary. Administration of LH-RH to adult male Skipper frogs resulted in marked hypertrophy and degranulation of basophils-2 (B2) in the pars distalis of the pituitary and a significant increase in their nuclear and cellular area. Concomitantly, there is a significant increase in the relative weight of the testes, in the number of cell nests containing secondary spermatogonia and primary spermatocytes, and in the nuclear diameter of the Leydig cells. There is also an increase in the Δ^5-3β-hydroxysteroid dehydrogenase and glucose-6-phosphate dehydrogenase activities in the Leydig cells. The results indicate that the B2 cells are gonadotrops and the hormone(s) secreted by B2 cells regulate the spermatogenetic and steroidogenic activity of the testis in *R. cyanophlyctis*.

Key words. LH-RH; frog testis; pituitary cytology; gonadotrops.

Administration of LH-RH affects the cytomorphology of gonadotrops and elevates the plasma levels of FSH and LH in mammals[2,3]. It has been shown that the pars distalis of the pituitary gland of the frog. *Rana catesbeiana* secretes two types of gonadotropins similar to mammalian FSH and LH, and administration of LH-RH causes an increase in the plasma levels of FSH and LH[4-6]. However, to the best of our knowledge, the effects of LH-RH on the cytomorphology of gonadotrops in amphibians have not been investigated. Though it has been assumed that the amphibian pituitary contains two types of gonadotrops similar to those of mammals[7] experimental studies have not yet convincingly proved this, and it is generally accepted that there is only one type of gonadotrop[8,9]. Since LH-RH specifically affects the release of gonadotropins, studies on the cytomorphological changes in the pituitary cell types following administration of LH-RH may help to find out whether there are two types of gonadotrops or not. Hence, the present study aims at investigating the effects of LH-RH on the cell types of the pars distalis with particular reference to gonadotrop(s) in the frog, *Rana cyanophlyctis*. Further, since the administration

Table 1. Effects of LH-RH on the mean nuclear diameter and length of different cell types in the pars distalis of *R. cyanophlyctis*

Groups and treatments	Mean nuclear diameter (µm ± SE) of Acidophils-1	Acidophils-2	Basophils-1	Basophils-2	Basophils-2
1) Controls (5)	4.95 ± 0.18	4.72 ± 0.09	4.40 ± 0.08	4.40 ± 0.06	4.60 ± 0.03
2) Frogs treated with LH-RH (5)	4.60 ± 0.13	4.61 ± 0.18	4.58 ± 0.09	5.48 ± 0.06	4.68 ± 0.09
	NS	NS	NS	$p < 0.05$	NS
	Mean length (µm ± SE)				
1) Controls	11.50 ± 0.48	9.12 ± 0.30	7.40 ± 0.19	11.28 ± 0.20	9.16 ± 0.20
2) Frogs treated with LH-RH (5)	10.98 ± 0.18	8.62 ± 0.38	7.86 ± 0.12	12.61 ± 0.21	9.41 ± 0.20
	NS	NS	NS	$p < 0.05$	NS

Number in parentheses indicates number of animals used. Means of control and experimental groups are compared using Student's t-test and judged significant if $p < 0.05$. NS = not significant.