of this hybrid with *R. lessonae*. This questions the status of one of the commonest European amphibians, accepted since Linneus as a *bona fide* species. Berger's results received substantial support from an electrophoretic investigation of blood proteins of a large group of frog material [4, 5]. The albumins of *R. ridibunda* and *R. lessonae* appear on the pherogramms as single bands but with different electrophoretic mobility, whereas *R. esculenta* shows in every case two albumin bands that correspond to the single bands of *R. ridibunda* and *R. lessonae*. However, the fact that two different types of double albumins are seen in *R. esculenta* could not be explained: in one case the two phases are distinctly divided and in the other they lie close together (Fig. 1a). The electrophoretic

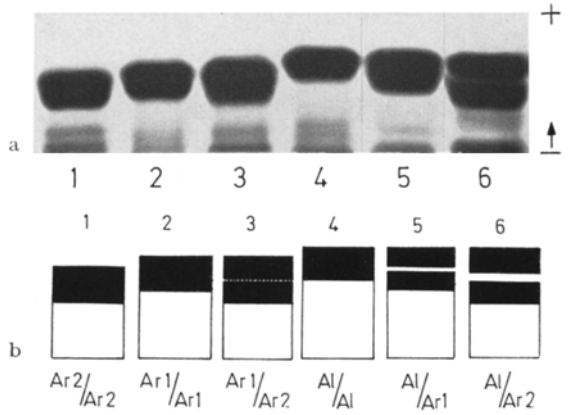

Fig. 1. a Electrophoretic patterns of the albumins of *Rana ridibunda* [1–3], *R. lessonae* [4] and the hybrid *R. esculenta* [5, 6] using 7,5% acrylamidegel. b Genetic differences in the albumins of the green frogs; drawn after Fig. 1a

methods were modified in the latest experiments on artificially produced hybrids [6], revealing that, in addition to the already known relatively slow moving albumin in *R. ridibunda*, there is a second albumin variation of even less electrophoretic mobility. Thus *R. ridibunda* has three possible albumin phenotypes, marked in Fig. 1b with the gene symbols Ar1/Ar1, Ar1/Ar2 and Ar2/Ar2. The albumin of *R. lessonae* appears to be very uniform (Al/Al). Consequently, the two albumin variations in *R. ridibunda* and the single band in *R. lessonae* are expressed in *R. esculenta* as two different double albumins, i.e. combinations of Al/Ar1 and Al/Ar2, respectively. Numerous populations of different European habitats have been investigated, and *R. esculenta* was found always to possess one of these double albumins, which leaves no doubt as to its hybrid nature. Apart from the nomenclatural consequences of the establishment of the hybrid status of *R. esculenta*, this frog is one of the animals most commonly used for experiments in European laboratories. A hybrid origin must now be taken into account. It is also apparent that in future greater importance must be accorded to hybridization as a mode of evolution within the animal kingdom, too.

Received July 5, 1973

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Prostaglandin-Releasing Effect of Exogenous Noradrenaline in Isolated Rabbit Heart

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Noradrenaline and prostaglandins are simultaneously released in response to stimulation of a sympathetic nerve [1]. The secretion of noradrenaline is reduced by exogenous prostaglandins of the E series and increased by the inhibition of prostaglandin synthesis caused by the 5,8,11,14-eicosatetraenoic acid or by indomethacin [2, 3]. It is suggested that locally synthesized prostaglandins could exert a modulating activity in sympathetic neurotransmission [4].

We are thus confronted with questions concerning the synthesis, release and mechanism of action of prostaglandins.

We therefore studied the prostaglandin-releasing effect of exogenous noradrenaline and the influence of α and β adrenergic receptors on the liberation of prostaglandins, using isolated rabbit hearts. The contractile force was measured using a strain gauge, and the heart rate by means of a Hoffner Beckman cardiotachymeter. These two parameters were simultaneously recorded on a Hoffner Beckman Dynograph. Perfusate was collected to determine prostaglandins: the pooled effluents from each heart were adjusted to pH 3 and extracted for lipids twice with equal volumes of ethyl acetate. The extracts were tested for smooth-muscle stimulating activity on a superfused rat-stomach strip.

Prostaglandins were determined in the effluents from:

1) spontaneously beating hearts (no drugs);

2) noradrenaline-treated hearts (10 µg/ml of noradrenaline infused over 15 min through a cannula immediately above the aorta at the rate of 1 ml/min and perfusate collected during the infusion);

3) hearts kept alive in Tyrode's solution containing either an α-blocking agent (phentolamine: $1 \cdot 10^{-6}\,M$) or a β-blocking agent (propranolol: $1 \cdot 10^{-6}\,M$), then infused with noradrenaline as in 2).

Under the experimental conditions adopted and with the method of determination used, no significant amount of prostaglandins was detected in the effluents from the untreated, spontaneously beating hearts. A 15-min infusion of noradrenaline (10 µg/ml/min) induced a total release of prostaglandins of 324 ± 56 ng ($n = 6$).

In the hearts previously treated with phentolamine or propranolol, the prostaglandins found were respectively 352 ± 63 ng ($n = 6$) and 359 ± 70 ng ($n = 6$). No statistically significant difference was found between these three series of experiments.

Exogenous noradrenaline induced a release of prostaglandins so that the experiments of Gilmore *et al.* were confirmed. However, the release of prostaglandins is not due to either post- or prejunctional adrenergic receptors since α- and β-blocking agents do not significantly affect their liberation.

Therefore it seems unlikely that prostaglandins could chemically mediate the control mechanism in the release of noradrenaline via α receptors [5].

Received June 18, 1973

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Immunosuppressive Activity of a Heat-Stable Factor Associated with Interferon

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Mouse interferon preparations derived from mouse brain and serum have been shown to exhibit antitumor activity [1]. Braun, Schizowa and Levy [2] have communicated that interferon preparations contain a factor that enhances antibody formation. This communication describes the presence of a heat-stable factor associated with mouse serum interferon which inhibits antibody synthesis and DNA synthesis in mouse leucocytes in the presence of phytohaemagglutinin (PHA).

Interferon was induced by a cell suspenison of *Brucella abortus* (Buck 19), inactivated at 60 °C for 1 hour. Suspensions containing 3 mg wet weight were injected i.p. and the blood was collected after 3 hours. Under these conditions the

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