

Cellular Location of Adrenergic Amines in Frog Sympathetic Ganglia

Modulation of transmission through sympathetic ganglia by an adrenergic inhibitory mechanism has been recently proposed¹. The observation that adrenergic nerves terminate on ganglionic neurones in a number of species of animals provides substantial support for this hypothesis².

Since frog paravertebral sympathetic ganglia are a convenient preparation for recording intracellularly from single ganglion cells, this preparation is being used to study the mechanism of adrenergic inhibition of ganglionic transmission. LIBET and TOSAKA³ have reported that slow inhibitory postsynaptic potentials can be recorded from frog ganglion neurones innervated by C fibres. Since they found that noradrenaline causes hyperpolarization of frog neurones, they suggest that adrenergic interneurons may cause the slow inhibitory potentials. However, the anatomical structures of frog and mammalian ganglion neurones are different, and the response of frog sympathetic neurones to adrenaline, noradrenaline and isoproterenol are different from the responses of mammalian ganglia⁴. Therefore, the cellular distribution of adrenergic

amines in frog paravertebral ganglia was investigated with a histofluorometric technique to see how the distribution compared with that of mammalian ganglia.

The paravertebral sympathetic ganglion chains from the seventh to tenth ganglia were removed bilaterally from 6 decapitated frogs, *Rana temporaria*. The tissue was frozen in propane, cooled in liquid nitrogen, freeze-dried, and treated with formaldehyde gas by a method previously reported⁵.

In sections of frog ganglia treated with formaldehyde gas for 3 h in order to visualize adrenaline, many cell bodies fluoresce strongly (Fig. 1). It is sometimes possible to see fluorescence extending into the axons leaving these neurones (Fig. 2). Although it is impossible to distinguish absolutely between noradrenaline and adrenaline with the method used, it is most probable that the frog ganglion neurones contain only adrenaline. The rate of development of fluorescence was similar to that for adrenaline, and adrenaline has been identified as the transmitter released from sympathetic nerves in the frog heart⁶.

Fluorescent adrenergic nerves terminating on sympathetic ganglion cell bodies or axons were never observed in any section from frog paravertebral sympathetic ganglia. This finding is in contrast to the observation that adrenergic nerves terminate on sympathetic neurones in mammalian paravertebral ganglia and occasionally terminate on neurones of mammalian paravertebral ganglia⁷. Hence these observations would suggest that the frog sympathetic ganglion preparation is not an appropriate one with which to investigate the mechanism of adrenergic inhibition of ganglionic transmission⁸.

Zusammenfassung. Die Lokalisation von Adrenalin in den sympathischen Ganglien (Frosch) wird mit Hilfe einer histo-fluorometrischen Methode untersucht. Es konnte Fluoreszenz in zahlreichen Zellen des Ganglions und in den aus den Zellen austretenden Axonen festgestellt werden. Fluoreszierende Nerven, die in Ganglion-Zellen eintreten, wurden nicht beobachtet, so dass die Fluoreszenz auf Adrenalin zurückzuführen sein dürfte.

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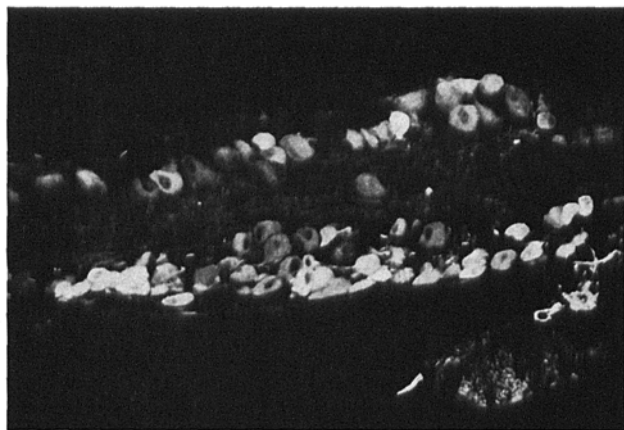


Fig. 1. Frog lumbar paravertebral ganglion treated with formaldehyde gas for 3 h to visualize adrenaline. Fluorescent adrenergic cell bodies of varying intensity are seen. $\times 120$.

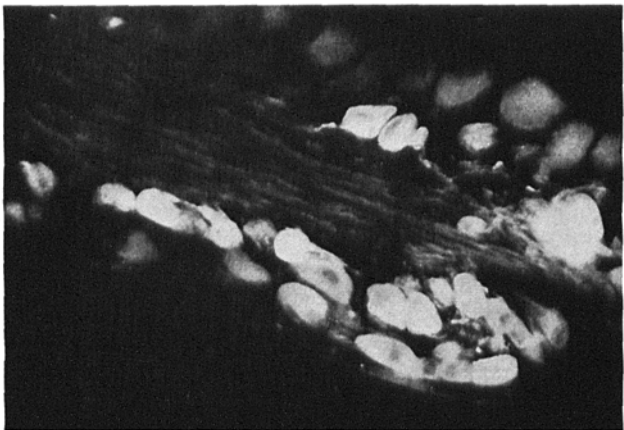


Fig. 2. Frog lumbar paravertebral ganglion treated with formaldehyde gas for 3 h to visualize adrenaline. Higher magnification. Fluorescent adrenergic cell bodies are seen and occasionally a strongly fluorescent axon leaving the cell body is observed. No nerves terminating on the ganglionic cells are seen. $\times 195$.

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- ⁸ The investigation has been supported by a grant from the Swedish Medical Research Council (No. B67-12-714-03) and Magn. Bergvalls Stiftelse, Stockholm, Sweden. The project was undertaken while one of the authors (R.M.) was visiting the Karolinska Institutet under a Special Fellowship N1-F3-GM-6506-01 from the National Institutes of Health (U.S.P.H.).
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