during GABA action. Thus, E_{GABA} seems close to the value for E_{Cl} (−42 mV) for ganglion cells calculated by Woodward, Bianchi & Erulkar (1969).

When external Cl^- was replaced with the impermeant anion isethionate, GABA depolarization was greatly enhanced (up to 50 mV) or hyperpolarization reversed to depolarization. Successive responses to GABA in isethionate solution waned rapidly, and were progressively restored on replacing Cl^-. This suggests that a brief application of GABA could produce a substantial net change in $[Cl^-]_i$ under these conditions.

We conclude that GABA increases Cl^- permeability in ganglion cells as in central neurones, and that ganglion depolarization occurs because $E_{Cl}<E_m$.

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Uptake of γ-aminobutyric acid (GABA) by sensory root ganglia

ZEHAVA GOTTESFELD, J. S. KELLY and F. SCHON* (introduced by L. L. IVERSEN)

Medical Research Council Neurochemical Pharmacology Unit, Department of Pharmacology, University of Cambridge

Brain slices (Iversen & Neal, 1968), sympathetic ganglia (Bowery & Brown, 1972) and retinae (Neal & Iversen, 1972) accumulate GABA by a high affinity uptake system, and autoradiographic studies suggest that this uptake occurs into both neurones and glial cells. Despite the lack of nerve terminals, a similar uptake of GABA has now been found in rat and cat sensory root ganglia.

Cat and rat ganglia were desheathed and incubated in oxygenated Krebs solution containing ^{3}H-GABA (0·1 μM). The Km value for GABA uptake of approximately 25 μm was similar to that in brain slices. As in the superior cervical ganglion the uptake of ^{3}H-GABA continued over a 4 h period. Amino-oxyacetic acid (AOAA) (10^{-5}M) added to the incubation media appeared to facilitate this uptake and tissue:medium ratios of 95:1 were achieved. Enhanced uptake also occurred in ganglia from animals pretreated with AOAA (40 mg/kg). Sensitive assay procedures showed the tissue of the ganglia to contain 0·26 μ moles/g tissue of GABA and a glutamic acid decarbovylase activity of (0·45 μ moles/g tissue)/hour. Similar levels were found in the ventral and dorsal roots by us in rats and by others in the cat.

Light microscopic autoradiography was carried out on glutaraldehyde fixed cat and rat ganglia following incubation in 80 μCi/ml ^{3}H-GABA (40 μM). Autoradiographs developed after a 7-day exposure period showed an intense uptake localized exclusively over satellite glial cells. The neuronal cell bodies, the remnants of the connective tissue sheath and the myelinated fibres were devoid of labelling.

When ganglia were incubated with 80 μCi/ml of ^{3}H-alanine or ^{3}H-glycine (80 μM) a 5-week exposure was necessary to obtain a similar silver grain density to that seen with ^{3}H-GABA after a one-week exposure. In contrast to GABA, both these amino acids were localized in neurones as well as in satellite glial cells. The active uptake of GABA in the sensory root ganglion is thus specific to this amino acid and exclusively localized in satellite glial cells.

Z.G. is on leave from the Weizmann Institute of Science while in receipt of an Israel Psychobiological Society award. F.S. is an MRC Scholar in the Physiological Laboratory, Cambridge.

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