the mode of action of substances causing neuronal depolarization. For instance, ouabaine, an inhibitor of Na^+-K^+-ATPase[18] in contrast to MBP, did not cause any shape change in platelets. This indicates that the MBP-induced shape change cannot be due to inhibition of Na^+-K^+-ATPase, and that the depolarizing effects of ouabaine[9] and of MBP in neuronal cells may well have different natures. Platelets might also be used to screen for substances able to antagonize the effects of MBP on membranes, which would be of potential interest for demyelinating diseases e.g. multiple sclerosis. In these disorders proteins seem to be liberated from the myelin sheaths, whose content of MBP, but not of histones, is quite high[19]. Indeed, increased amounts of MBP-like material have been found in the cerebrospinal fluid of patients with demyelinating disorders[20-22]. If these proteins were to act on neuronal membranes this might cause neurological manifestations e.g. during episodes of acute exacerbation of the disease. Finally, it may be of interest to investigate whether there is a difference between platelets of patients with demyelinating disorders and those from healthy controls in their reaction to MBP.

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Effect of the polychlorinated biphenyl preparation, Aroclor 1242, on the quantity of neurosecretory material in the medulla terminalis X-organ of the fiddler crab, *Uca pugilator*[1]

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Summary. Exposure of the fiddler crab, *Uca pugilator*, to the PCB preparation, Aroclor 1242, produces an increase in the quantity of neurosecretory material in the medulla terminalis X-organ. This Aroclor apparently inhibits release, but not synthesis, of one or more neurohormones.

Polychlorinated biphenyls (PCBs) are extremely persistent, globally distributed pollutants[3]. Many animals, including the fiddler crab, *Uca pugilator*, the species used in this investigation, have been found to accumulate PCBs in their tissues[4]. Exposure of this crab to the PCB preparation, Aroclor 1242, results in decreased dispersion of the pigment in its melanophores[5]. Those crabs exposed to Aroclor 1242 had up to 4 times more melanin-dispersing hormone (MDH), a neurohormone, in their eyestalks than did control crabs. The PCB preparation appeared to inhibit MDH release but not its synthesis. In the eyestalk of the fiddler crab, MDH is released from the sinus glands. These glands are neurohemal organs. Most, if not all, of the neurosecretory axons that enter a sinus gland originate in the medulla terminalis X-organ of each eyestalk. In a recent study of the eyestalk of the crayfish, *Orconectes virilis*, the investigators could find no evidence that neurosecretory axons came from any other area than the medulla terminalis X-organ[6]. The object of this investigation was to determine whether cytological evidence of an effect of Aroclor 1242 on the neuroendocrine system of the fiddler crab could be obtained.

Materials and methods. Mature female specimens of *Uca pugilator*, having a carapace width of 1.5–1.6 cm, from the area of Panacea, Florida, were used. At noon, crabs were selected from the stock supply and placed in white enameled pans (18 cm diameter) containing artificial sea water (Instant Ocean, Aquarium Systems) alone, and placed under a constant illumination of 2100 lux at 24 °C. 24 h later those crabs whose melanophores were at Hogben-Slome stage 3 were selected for further use[7]. According to the Hogben-Slome scheme stage 3 represents an intermediate degree of pigment dispersion with stage 1 representing maximal concentration of the pigment and stage 5 maximal dispersion. The selected crabs were then divided into 2 groups and placed into white enameled pans which contained either 8 ppm Aroclor 1242 (Monsanto Lot Number G266K) in a solution of 0.1% acetone in artificial sea water or only 0.1% acetone in artificial sea water. The Aroclor had first been dissolved in acetone which explains the presence of acetone in both the experimental and control containers. The volume of liquid in each pan was 400 ml. The 2 groups of crabs were again exposed to the constant illumination of 2100 lux at 24 °C for 24 h after which their melanophore stages were again determined and their eyestalks were removed. The internal tissues of both eyestalks from 5 experimental and 5 control crabs were then dissected out, fixed in Bouin's solution, embedded in paraffin, cut into longitudinal serial sections 8 µm thick, and stained with Gomori's chrome-alum hematoxylin and

Neurosecretory activity in the medulla terminalis X-organ

	Crab number	Staining index
PCB-treated	1	2
	2	3
	3	2
	4	3
	5	3
		Average 2.6*
Control	1	1
	2	1
	3	2
	4	2
	5	1
		Average 1.4

*Significantly greater than control ($p < 0.05$).

phloxin[8]. The secretory activity of the neurosecretory cell bodies in the medulla terminalis X-organ of the sectioned eyestalks was then evaluated by using the following index numbers to stage the cells: 0, neurosecretory granules absent; 1, very few granules; 2, intermediate between 1 and 3; 3, a large number of granules. This system is essentially that of Matsumoto, but he numbered his stages 0, 1, 3, and 5[9]. A cell whose cytoplasm is crowded with neurosecretory granules presumably contains more neurohormone than does a cell with few or no granules. Statistical evaluation of the data was performed using the Student's t-test.

Results and discussion. When the eyestalks were removed, the mean melanophore stage of the crabs that had been in the acetone-sea water for 24 h was 3.4 whereas that of the crabs in the PCB preparation was 1.7. This difference (3.4 versus 1.7) and the decrease from the initial melanophore stage (1.7 versus 3.0) were not only statistically significant ($p < 0.01$ for both) but were also consistent with the previous observation that Aroclor 1242 prevents the pigment in the melanophores from remaining as dispersed as in control crabs[5]. The slightly increased level of melanin dispersion, up from the original 3.0 to 3.4, in the control crabs was presumably due in large measure to the continuous exposure to the bright illumination (2100 lux) in contrast to the 422 lux the crabs had been exposed to in the stock tank prior to the start of the experiment. Bright illumination induces melanin dispersion in the fiddler crab[10].

Examination of the eyestalk sections revealed that the quantity of neurosecretory material in the neurosecretory cell bodies in the medulla terminalis X-organs of the crabs exposed to the PCB preparation was significantly greater than in the control crabs (table). This observation is consistent with the previously obtained data which showed that eyestalks of crabs exposed to Aroclor 1242 contained more MDH than did eyestalks of control crabs[5]. Of course, all of the neurosecretory granules stained were not MDH-containing alone. The eyestalk secretes several different neurohormones[11]. Additional experiments are being performed to determine what eyestalk neurohormones in addition to MDH are also affected by PCBs.

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Glomerular filtration rate and kidney blood flow in alloxan and streptozotocin diabetic rats

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Summary. Glomerular filtration rate (GFR), cardiac output, regional blood flow and kidney weight were measured in alloxan and streptozotocin diabetic rats at different times after the administration of diabetogen. A high GFR was found together with increased kidney weight and reduced blood flow.

The role of diabetic nephropathy as a cause of death in late diabetes mellitus underlines the importance of studying changes in renal structure and function early in the disease. A measurable thickening of the basement membrane and mesangial region has been clearly shown in recent studies using the electron microscope. It takes place long before the advent of any clinical evidence of nephropathy[1-3]. It is thought that this thickening is the forerunner of more advanced disease which leads to renal failure.

Recent clinical studies are agreed in showing an increase in the glomerular filtration rate (GFR) in early diabetes[4-7]. This elevation of GFR is accompanied by normal or slightly increased renal plasma flow (RPF) in some reports[5,6] and reduced RPF in others[4]. The exact mechanism behind this high GFR is not known. It has been variously suggested that it might be related to an increase in RPF, a decrease in RPF and hence a higher filtration pressure or to an increase in the permeability of the glomeruli. Increased kidney size was also reported to be behind this abnormality[8,9].

All the previously mentioned studies were carried out on human beings where ethical considerations limit the nature of the investigations capable of being executed. In particular, simultaneous estimation of cardiac output and renal blood flow, while extremely difficult in humans, is quite feasible in experimental animals, using the labelled microsphere technique previously described[10].

Previous work on experimental diabetes has been mainly directed to the toxic effects of the diabetogenic agents on the kidney itself. Alloxan and streptozotocin were both reported to cause renal lesions[11-14]. However, these lesions were also shown to be due to the diabetic condition or to