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Phil. Trans. R. Soc. Lond. B 1985 **308**, 409

doi: 10.1098/rstb.1985.0043

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3. Effects of capsaicin on nociceptive afferents from the skin

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Capsaicin is a well-known excitant of cutaneous C-fibre nociceptors. In addition it has long-lasting effects on the functions of C-afferents in the rodent. Administration of capsaicin to neonatal rats causes a large reduction in the number of all functional classes of afferent C-fibre (Carpenter & Lynn 1983; Lynn 1984). We have now examined the effects of direct application to cutaneous nerves in adult animals and have found dramatic and selective effects on axonal conduction and nociceptor function.

In anaesthetized adult rats, capsaicin (1% in olive oil) rapidly blocks conduction in over 90% of C-fibres and about 25% of A-fibres when applied directly to the intact saphenous nerve (Pini 1983). Similar effects on C-fibre conduction have also been reported by Petsche *et al.* (1983). Conduction resumes in all A-fibres and about 30% of C-fibres within 1 h of removing the capsaicin. However, axons of many polymodal nociceptor units remain blocked for 1–7 days. No comparable long-term block occurs in the axons of other types of C-afferent (e.g. sensitive mechanoreceptors and thermoreceptors). Similar immediate effects on nerve conduction have not been found in experiments on the sural nerve of the rabbit.

To study nociceptor function, single afferent units have been isolated from the saphenous nerve of rats by microdissection. Fourteen to 19 days after a 30 min capsaicin application, samples of C-units isolated contained only 21% polymodal nociceptors compared with 68% in control, vehicle-treated, nerves. Conversely, the proportion of axons that were excited by electrical stimulation of the nerve, but could not be excited from the skin, was raised from 25% in controls to 77% after capsaicin. It therefore appears that approximately 70% of polymodal nociceptor terminals in the skin have become inexcitable (Lynn & Pini 1984). We now have preliminary evidence that at four months following this treatment a considerable number of polymodal nociceptor axons have degenerated since C-compound action potentials are much reduced and few single C-polymodal nociceptor units can be isolated.

Thus direct application of capsaicin to rodent nerves alters the function of C-afferents, especially the polymodal nociceptors. How far these effects occur in non-rodent mammals remains to be established.

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