Ototoxicity of salicylate is accompanied by a temporary hearing loss and tinnitus and has therefore been used to study tinnitus in animal models. Salicylate induced elevated central auditory activity has been interpreted as a correlate of tinnitus. Whether this elevated activity in the central auditory system is due to an increased activity in the auditory nerve is still under discussion. To explore this issue, we recorded the activity of single auditory nerve fibres in anaesthetised gerbils following systemic injection of salicylic acid.

Firstly, compound action potential (CAP) thresholds were determined at 5-0 min intervals. Fifteen to 30 min after 200 mg/kg salicylic acid, threshold loss developed in the high frequency range. At 2 h CAP threshold loss reached a plateau amounting to 15-20 dB above 16 kHz, 0-5 dB below 2 kHz. An almost immediate start of threshold loss was observed after 400 mg/kg salicylic acid. A plateau of threshold loss was reached after 1.5 h with values of 25 dB in the high, 5-10 dB in the low frequency range.

Secondly, responses of single auditory nerve fibres were studied after administration of 200 mg/kg salicylic acid. Frequency tuning curves and rate intensity (RI) functions at characteristic frequency (CF) were measured. Two hours and more after application, single fibre thresholds were elevated by about 20 dB at all CFs. Sharpness of tuning was reduced. Mean spontaneous rate was significantly reduced at CFs below 5 kHz (mean: 44 vs 28 AP/s). At CFs
above 5 kHz mean spontaneous rate remained unchanged. In RI functions no change in maximum discharge rate was observed. The altered response properties can be interpreted by the known effects of salicylate on the prestin mediated active process of the outer hair cells. The elevated activity in the central auditory system after salicylate intoxication thus cannot be caused by cochlear nerve hyperactivity.