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Nerve excitability and structural changes in myelinated axons from diabetic mice.

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OBJECTIVE:

The mechanisms associated with nerve dysfunction and axonal loss in diabetes has not been fully clarified. Excitability and pathological aspects in nerves from diabetic mice were studied in order to explore the pathophysiology of diabetic neuropathy.

METHODS:

Myelinated nerve fibres from the sciatic nerve of BKS.Cg-m (+/+) Lepr (db) /J mice were studied by registering the CMAP controlled by an automated threshold tracking method. The sciatic nerve was also studied pathologically.

RESULTS:

Diabetic mice displayed longer latencies, higher thresholds and lower amplitudes compared to controls and had a rightward shift in the stimulus response curves. Strength-duration time constant was lower in diabetic mice but not reaching statistical significance ($p=0.09$). Diabetics displayed an increase in accommodation, with a smaller change in excitability in threshold electrotonus. Refractoriness, mean superexcitability and late subexcitability were reduced in diabetic mice. Diabetic mice had a larger number of myelinated fibres compared to controls ($p<0.05$), but larger than 9 μm were virtually absent, accounting for near 7% in control animals.

CONCLUSIONS:

Db/db mice develop electrophysiological changes suggestive of membrane depolarization as the result of Na(+)/K(+) pump impairment. Loss of large myelinated fibres might also contribute to the nerve excitability profiles in this model.