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Regenerative failure of diabetic nerves bridging transection injuries.

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Abstract

BACKGROUND: Failed regeneration compounds the deficits imposed by diabetes from peripheral neuropathy. In this work, we addressed how diabetes or local glucose toxicity might impact peripheral nerve trunk regeneration and reconstitution across major sciatic nerve transection injuries of rats.

METHODS: Specific conduits, amendable to manipulation of infused glucose concentrations through a T connection, were perfused with 5 or 30 mmol/L glucose in nondiabetics or 5 mmol/L glucose in rats with experimental diabetes. Quantitative early and later regenerative outgrowth was measured.

RESULTS: Local glucose exposure had no impact on early axon or Schwann cell outgrowth or partnering nor later myelinated axon regeneration. Despite only mildly attenuated early sprouting of axons with Schwann cells, diabetic bridges exhibited massive later failure of reconstitution by 3 weeks after injury.

CONCLUSION: Diabetes is associated with severe limitations in regenerative success, despite appropriate early axon outgrowth