

# Neural control of the expression of a Ca<sup>2+</sup>-activated K<sup>+</sup> channel involved in the induction of myotonic-like characteristics

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1. Expression of the apamin-sensitive K<sup>+</sup> channel (SK<sup>+</sup>) in rat skeletal muscle is neurally regulated.

The regulatory effect of the nerve over the expression of some muscle ion channels has been attributed to the electrical activity triggered by the nerve and/or to a trophic effect of some molecules transported from the soma to the axonal endings. 2. SK<sup>+</sup> channels apparently are involved in myotonic dystrophy (MD), therefore understanding the factors that regulate their expression may ultimately have important clinical relevance. 3. To establish if axoplasmic transport is involved in this process, we used two experimental approaches in adult rats: (a) Both sciatic nerves were severed, leaving a short or a long nerve stump attached to the anterior tibialis (AT), (b) Colchicine or vinblastine (VBL), two axonal transport blockers of different potencies, was applied on one leg to the sciatic nerve. To determine whether electrical activity affects the expression of SK<sup>+</sup> channels, denervated AT