NF-?B activation by depolarization of skeletal muscle cells depends on ryanodine and IP3 receptor-mediated calcium signals

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Depolarization of skeletal muscle cells by either high external K + or repetitive extracellular field potential pulses induces calcium release from internal stores. The two components of this release are mediated by either ryanodine receptors or inositol 1,4,5-trisphosphate (IP3) receptors and show differences in kinetics, amplitude, and subcellular localization. We have reported that the transcriptional regulators including ERKs, cAMP/Ca2+-response element binding protein, c-fos, c-jun, and egr-1 are activated by K+-induced depolarization and that their activation requires IP3-dependent calcium release. We presently describe the activation of the nuclear transcription factor NF-?B in response to depolarization by either high K+ (chronic) or electrical pulses (fluctuating). Calcium transients of relative short duration activate an NF-?B reporter gene to an intermediate level, whereas long-lasting calcium increases obtained by prolonged electrical stimulation protocols of various freque