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Nitric Oxide Synthase In Skeletal Muscle Repair Following Strain Injury

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Following muscle injury, satellite cells are recruited and activated to enable muscle repair. Previous studies using nitric oxide synathase (NOS) inhibition, *mdx* dystrophy mice and NOS-I knock-out mice have suggested that nitric oxide release may mediate satellite cell activation. **PURPOSE:** To assess the presence of NOS in strain-induced muscle injury in conjunction with two muscle regulatory factors: MyoD and myogenin.

METHODS: Using female Sprague Dawley rats, muscle strains were produced by manually stretching (50 repetitions) an activated plantar flexor group through its normal range of motion. Muscle biopsy samples were evaluated 48- hours after strain injury for the presence of nitric oxide synthase, MyoD, and myogenin using Western immunoblot analysis. Transcript levels for neuronal (n) NOS, endothelial (e) NOS, inducible (i) NOS, MyoD and myogenin were assessed using reverse transcriptase polymerase chain reaction (RT-PCR) and kinetic PCR. **RESULTS:** An increase in myogenin transcripts and a decrease in nNOS transcript expression was observed 48- hours post-strain injury by RT-PCR. Western immunoblot analysis revealed increased eNOS, MyoD, and myogenin protein levels 48- hours post-strain injury, but a decrease in nNOS protein levels. **CONCLUSION:** NOS has been reported to play a role in muscle repair. Although nNOS expression was decreased, eNOS expression increased providing support for other NOS isoforms in muscle repair.

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