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Different magnitude of resistance to non-depolarizing muscle relaxants in dexamethasone-treated rat diaphragm associated with altered acetylcholine receptor expression

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ABSTRACT. The aim of this study was to investigate the influence of chronic dexamethasone (Dex) administration on rat diaphragm sensitivity to non-depolarizing muscle relaxants (NDMRs) and muscular nicotinic acetylcholine receptor (nAChR) expression, which may help direct future administration of NDMRs. Adult male Sprague-Dawley rats were randomized to receive a daily intraperitoneal injection of Dex (600 μg/kg body mass) or an equivalent volume of saline (N = 20 in each group) for 14 days. We evaluated isometric twitch tensions of nerve-hemidiaphragm preparations elicited by indirect supramaximal stimulation at 0.1 Hz. Real-time quantitative PCR was performed to determine the mRNA expression of two nAChR subunits (ε-subunit and γ-subunit) in the diaphragm. Dex administration markedly ($P < 0.01$) increased the 50% twitch depression (IC_{50}) of the three NDMRs. The IC_{50} ratio, which standardized the magnitudes of the resistance, was the

largest for atracurium, with the second largest for vecuronium and the smallest for rocuronium ($P < 0.01$). The ε- and γ-subunit mRNAs were both upregulated with an increased γ/ε ratio in rats exposed to Dex. The results indicated that chronic Dex administration induces hyposensitivity to NDMRs, the degree of which depends on the kind of neuromuscular blocker, and is associated with increased nAChR expression.

Key words: Glucocorticoids; Neuromuscular blocking agents; Acetylcholine receptor; Hemidiaphragm

