stimulus. In contrast, the peak to peak amplitude of the EMG activity (Mmax) did not show any significant change after the fatiguing contraction. Muscle activation, estimated by the interpolated-twitch method, was maximal in control conditions but also after the fatigue test.

In conclusion, the absence of significant change in muscle activation during an MVC after fatigue suggests that the central neural drive is not impaired and that peripheral mechanisms play a key role in the observed force decrease. The similar reduction in the rate of torque development during voluntary ballistic and electrically-induced contractions, also indicates that peripheral mechanisms are mainly responsible for the reduction in muscle speed during ballistic contractions. At peripheral level, the absence of significant change in Mmax amplitude indicates that failure in neuromuscular transmission and sarcoplemmal excitation cannot explain the reduction in muscle force and contractile kinetics. This observation, associated with a decrease in mechanical twitch amplitude, suggests that alterations of processes located beyond the neuromuscular junction and muscle membrane, possibly involving the excitation-contraction coupling, should be responsible for the fatigue-related reduction in maximal force and rate of torque development during ballistic contractions.

Keywords: fatigue, ballistic contraction, EMG