IMATINIB MESILATE (GLEEVEC®) AMELIORATES THE DYSTROPHIC PHENOTYPE IN *MDX* MICE

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Duchenne Muscular Dystrophy (DMD) is a neuromuscular disorder caused by dystrophin gene mutations. It is characterized by progressive skeletal muscle degeneration that leads to weakness and early death by respiratory and cardiac breakdown. There is no specific treatment to DMD. Preclinical tests to find new drugs that can stop/retard DMD evolution are usually performed in exercised *mdx* mouse. One important feature in DMD is the massive muscle infiltration by immune cells and the replacement by fibrous or fatty tissue. Immunomodulators have been recently emerged for DMD trials. Imatinib mesilate is a specific inhibitor of tyrosine kinases, such as Bcr-Abl, PDGFR-β and c-Kit receptors. It also inhibits the profibrogenic activity of TGF-β. The present study aimed to evaluate imatinib mesilate in *mdx* mice submited to treadmill exercise. Comparative analyses showed that 0,125 mg/mice/day resulted in amelioration of the muscular conditions, increased force increment and decreased CK levels. Histological analyses of the gastrocnemius showed abrupt decreasing of the area occupied by injured myofibers (2.50±1.21% against 19.91±6.62% in untreated *mdx*), while diaphragm showed no significant difference. Taken together, these data suggest that Gleevec® can retard the deleterious effects in DMD patients, and could be used as potential drug to future clinical tests.