Annexin A1 is a biomarker of T-tubular repair in skeletal muscle of non-myopathic patients undergoing statin therapy

ID 2833284
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Year 2013
Title Annexin A1 is a biomarker of T-tubular repair in skeletal muscle of nonmyopathic patients undergoing statin therapy
Journal The FASEB Journal
Volume 27
Number 6
Pages / **Article-Number** 2156-64
Keywords plasma membrane repair, Ca2+-dependent translocation, dysferlin
Skeletal muscle complaints are a common consequence of cholesterol-lowering therapy. Transverse tubular (T-tubular) vacuolations occur in patients having statin-associated myopathy and, to a lesser extent, in statin-treated patients without myopathy. We have investigated quantitative changes in T-tubular morphology and looked for early indicators of T-tubular membrane repair in skeletal muscle biopsy samples from patients receiving cholesterol-lowering therapy who do not have myopathic side effects. Gene expression and protein levels of incipient membrane repair proteins were monitored in patients who tolerated statin treatment without myopathy and in statin-naive subjects. In addition, morphometry of the T-tubular system was performed. Only the gene expression for annexin A1 was up-regulated, whereas the expression of other repair genes remained unchanged. However, annexin A1 and dysferlin protein levels were significantly increased. In statin-treated patients, the volume fraction of the T-tubular system was significantly increased, but the volume fraction of the sarcoplasmic reticulum remained unchanged. A complex surface structure in combination with high mechanical loads makes skeletal muscle plasma membranes susceptible to injury. Ca(2+)-dependent membrane repair proteins such as dysferlin and annexin A1 are deployed at T-tubular sites. The up-regulation of annexin A1 gene expression and protein points to this protein as a biomarker for T-tubular repair.
Publisher FEDERATION AMER SOC EXP BIOL
ISSN/ISBN 0892-6638
edoc-URL http://edoc.unibas.ch/dok/A6338586
Full Text on edoc No;
Digital Object Identifier DOI 10.1096/fj.12-219345
PubMed ID http://www.ncbi.nlm.nih.gov/pubmed/23413360
ISI-Number WOS:000319667600007
Document type (ISI) Journal Article