Evidence has been obtained that reactive oxygen (ROS) and nitrogen species (RNS) are formed in skeletal muscle cells during contractions. However, the effect of long chain fatty acids (LCFA) on ROS and RNS production in skeletal muscle cells remains unknown. In this study, the acute and chronic effect of palmitic acid (PA) on superoxide (O$_2^-$) as well as RNS production by skeletal muscle cells in culture under spontaneously contractions was investigated. Muscle cells were acutely (1 h) or chronically (24, 48 and 72 h) incubated at 37° C in DPBS containing PA (25 61549;M) and cytochrome c (50 61549;M) as O$_2^-$ detector. Nitrite/nitrate was determined by Griess method and NO by DAF-DA assay. DPI (2 61549;M) and allopurinol (600 61549;M) were added as NADPH-oxidase and xanthine-oxidase inhibitor, respectively. Whereas, CCCP (10 61549;M) was added as mitochondrial uncoupling. PA increased basal O$_2^-$ production (p < 0.05), whereas DPI abolished this effect (p > 0.05 compared to control). Accordingly, Western blotting of p47, a cytosolic NADPH-oxidase complex subunit, exhibited elevated phosphorylation level in the presence of PA. Allopurinol nor mitochondrial uncoupling CCCP had a significant effect on O$_2^-$ production (p > 0.05). In contrast, during chronic PA treatment, cultured muscle cells exhibited a significant decrease of superoxide production, suggesting that a prolonged exposure to PA might upregulate protective mechanisms such as an increasing UCP-3 expression. Similarly, PA increased both nitrite/nitrate and NO production in skeletal muscle cells, indicating an elevation in NO production (p < 0.05). In addition to NO production, western blotting analysis showed that PA induced an increase in protein nitration. Our findings show that ROS and RNS production is modulated by elevated availability of fatty acids in skeletal muscle cells. 

Keywords: Muscle, Fat Metabolism, Free Radicals