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Oxidative stress, more precisely the damage caused by an abnormal production of reactive oxygen species (ROS), is a key mechanism in ALS pathogenesis. NADPH oxidases are a family of enzymes responsible for the production of ROS. In our previous study, an increased survival of ALS-affected individuals has been related to low activity of NADPH oxidase-2 (NOX2) in their neutrophils (1). Based on this finding, this work aimed to evaluate the protein levels of NOX2 in neutrophils of patients affected by ALS, in order to verify a possible correlation between levels and activity.
The analysis was performed in a cohort of 40 subjects using Western immunoblot technique. The attention was mainly focused on ALS patients carrying mutations of copper-zinc superoxide dismutase-1 (SOD1) gene, since mutations of SOD1 are referred to alter NOX2 activity (2).

(1) Marrali G et al. NADPH oxidase (NOX2) activity is a modifier of survival in ALS. J. Neurol. 2014
(2) Harraz et al. SOD1 mutations disrupt redox-sensitive Rac regulation of NADPH oxidase in a familial ALS model. J Clin Invest. 2008