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SYMPOSIUM 9 - OTHER TOPICS: BASIC RESEARCH

55ASM-0001 FT | Analysis of Becline-1, VPS34, LC3, and p62 in T-Lymphocytes of patients with Systemic Lupus Erythematosus

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Background: Dysregulation of autophagy has been implicated in numerous autoimmune diseases. Several lines of evidence are emerging to support the role of autophagy in progression and pathogenesis of systemic lupus erythematosus (SLE). The aim of this study is to analyze the expression of key autophagy proteins in T-lymphocytes of patients with SLE.

Materials and Methods: Western blot analysis was used. Two groups were compared using Student's test. At the first stage was carried out an analysis of Beclin-1, which is responsible for the initiation of autophagy. There is a significant increases of it in SLE group compared with the control. The same result was obtained in the analysis of Vps34. Beclin-1 and Vps34 are part of the complex, which gives rise to the formation of autophagosomes. p62 is a central marker of autophagic degradation and plays a role in the induction of autophagy. We showed that the expression of p62 in SLE is higher. At the final stage, the content of LC 3 was determined. The levels of LC3-I and LC3-II in SLE were significantly higher than in healthy donors. And the ratio of LC3-I/LC3-II was lower.

Results: We decided to analyze the content of p62, LC3-I, LC3-II and LC3-I/LC3-II ratio depending on the severity of the disease: acute, subacute and chronic. The most significant was an increase in p62 and LC3-II and a decrease in the ratio LC3-I/LC3-II in the group with chronic SLE.

Conclusions: The results indicate autophagy disorders in SLE. The high level of p62 and LC3-II indicate lack fusion of autophagosomes with lysosomes. And incomplete autophagy leads to the accumulation of large autophagosome numbers inside the cell and more severe disease.

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