



JAK2-STAT3 Pathway Mediated Satellite Cell Apoptosis to Govern Skeletal Muscle Growth with Lysine

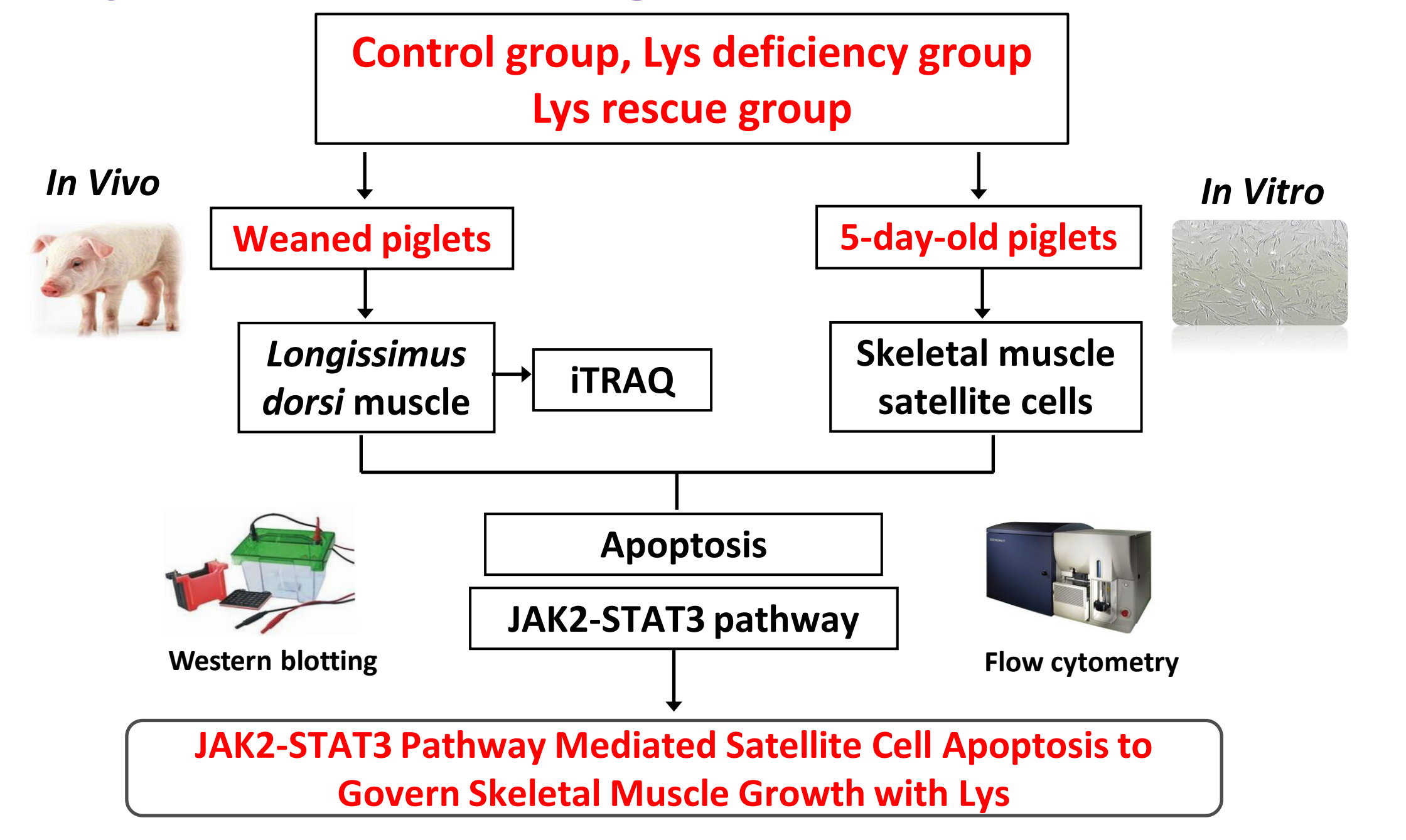
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Introduction

Apoptosis is programmed cell death that can be stimulated by external stress or nutrition restrictions. Lysine (Lys) is an essential amino acid for pig growth, and the relationship between Lys deficiency caused apoptosis and inhibition of skeletal muscle growth remains unknown. The objective of this study was to investigate whether apoptosis could be regulated by Lys supplementation and the potential mechanism.

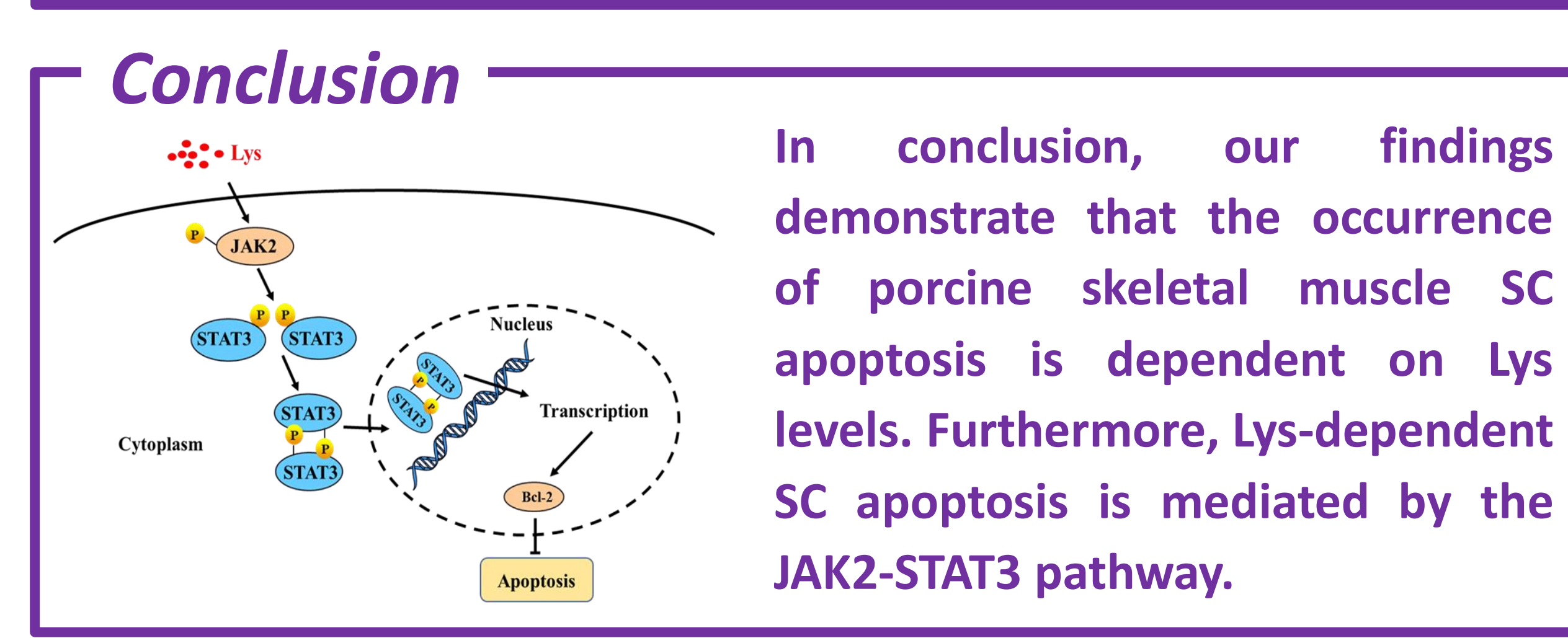
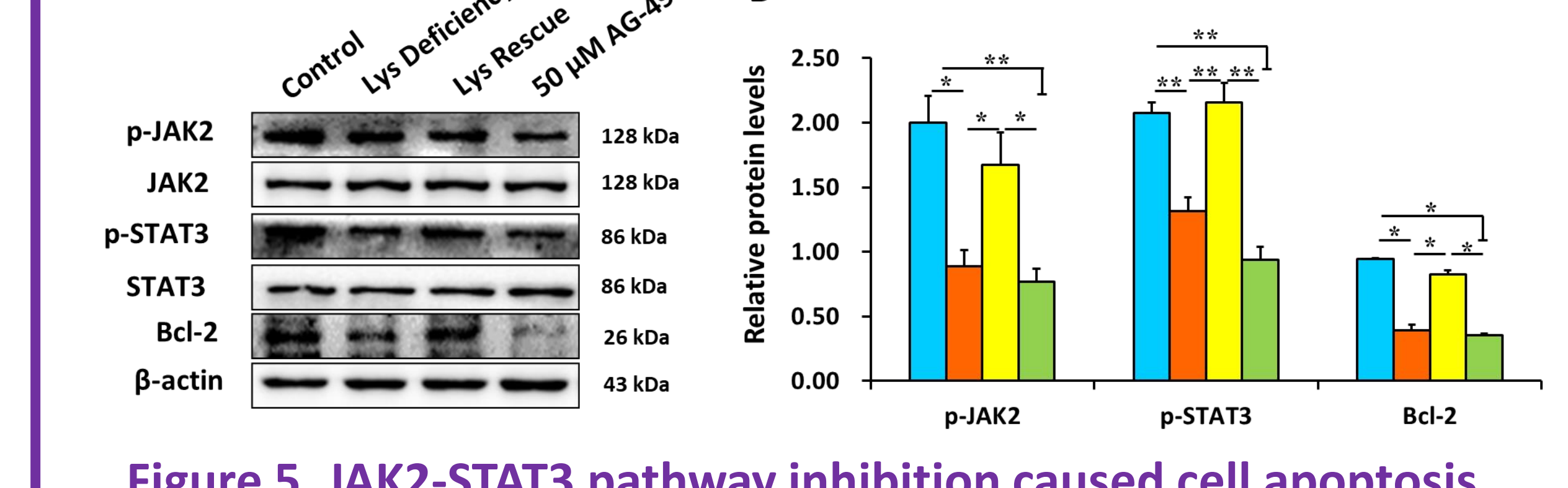
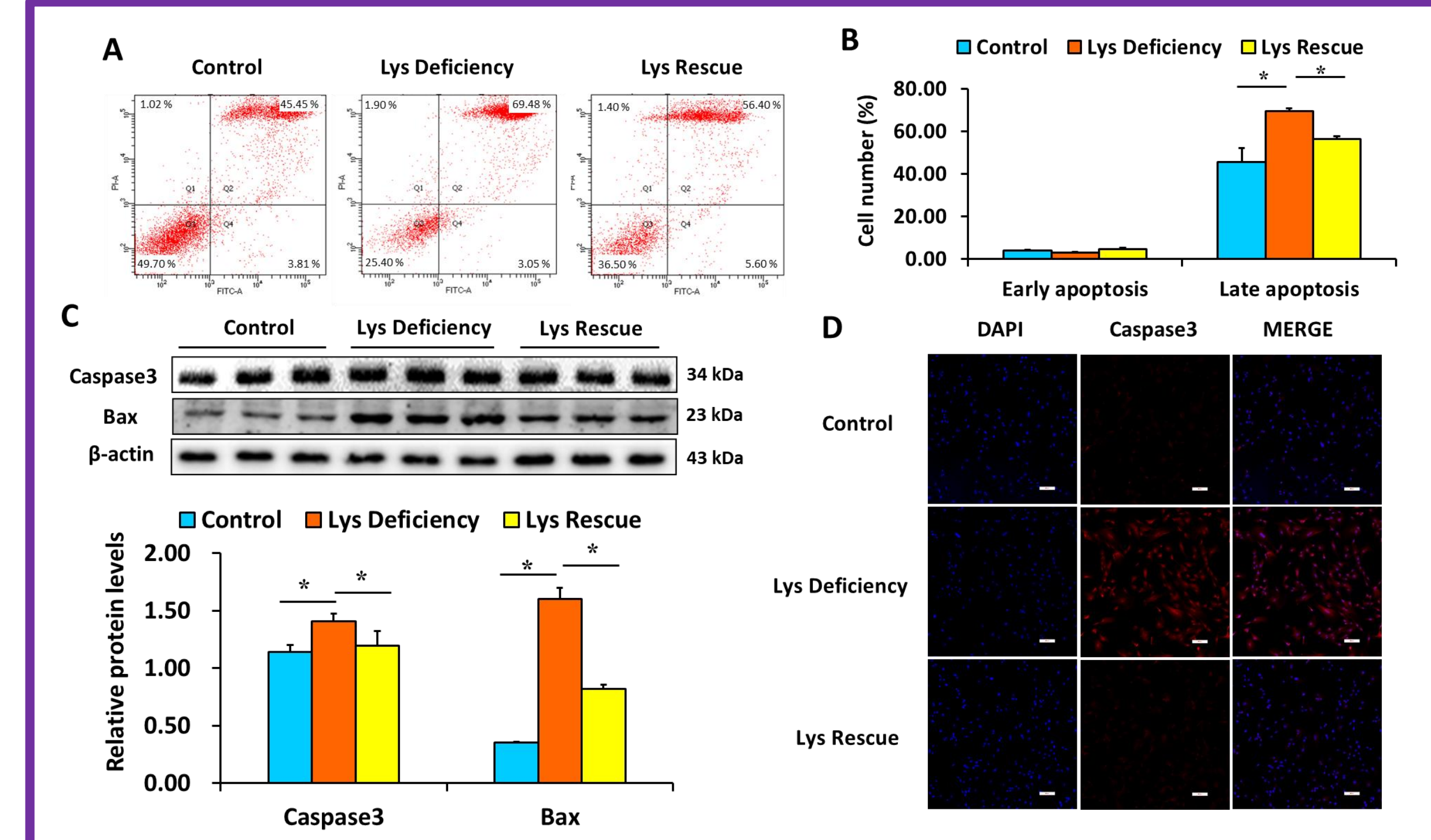
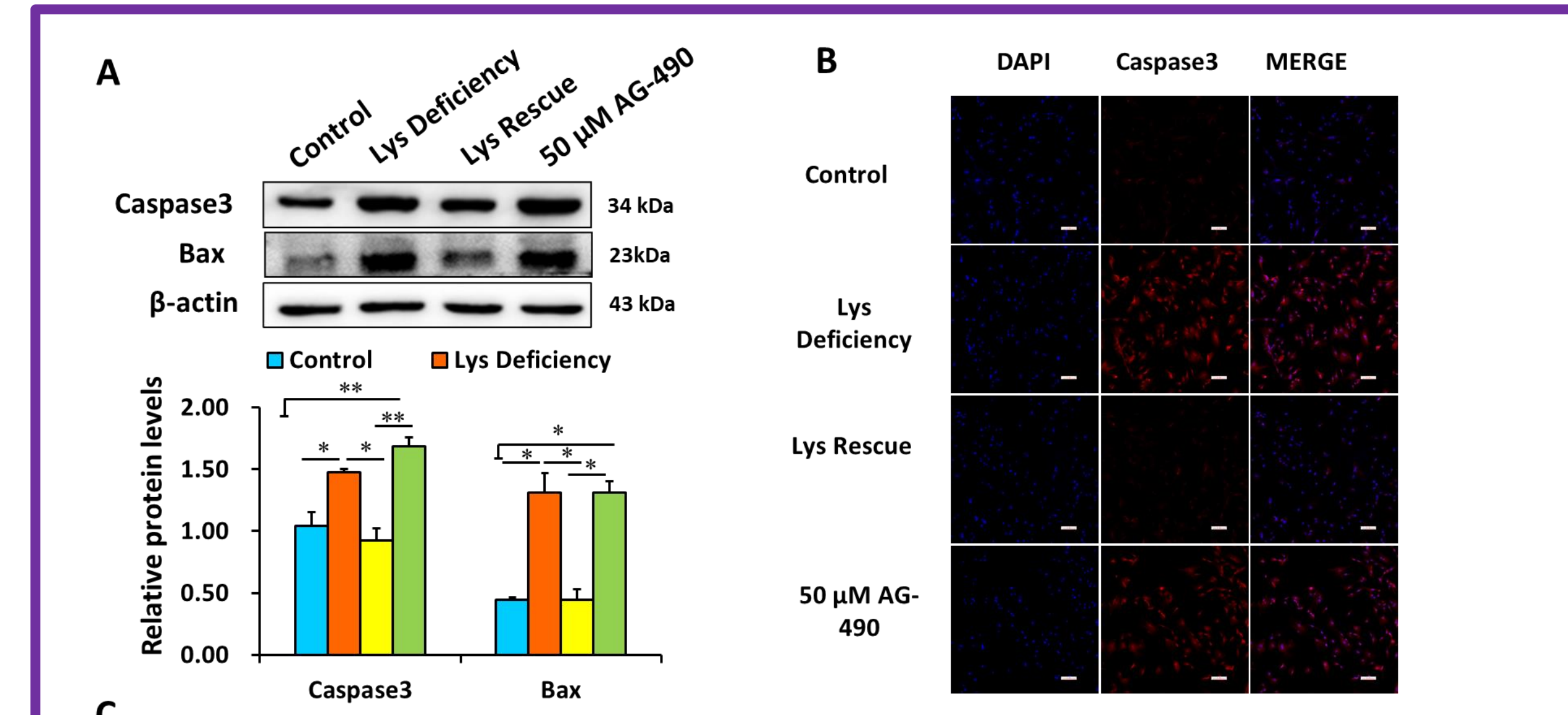
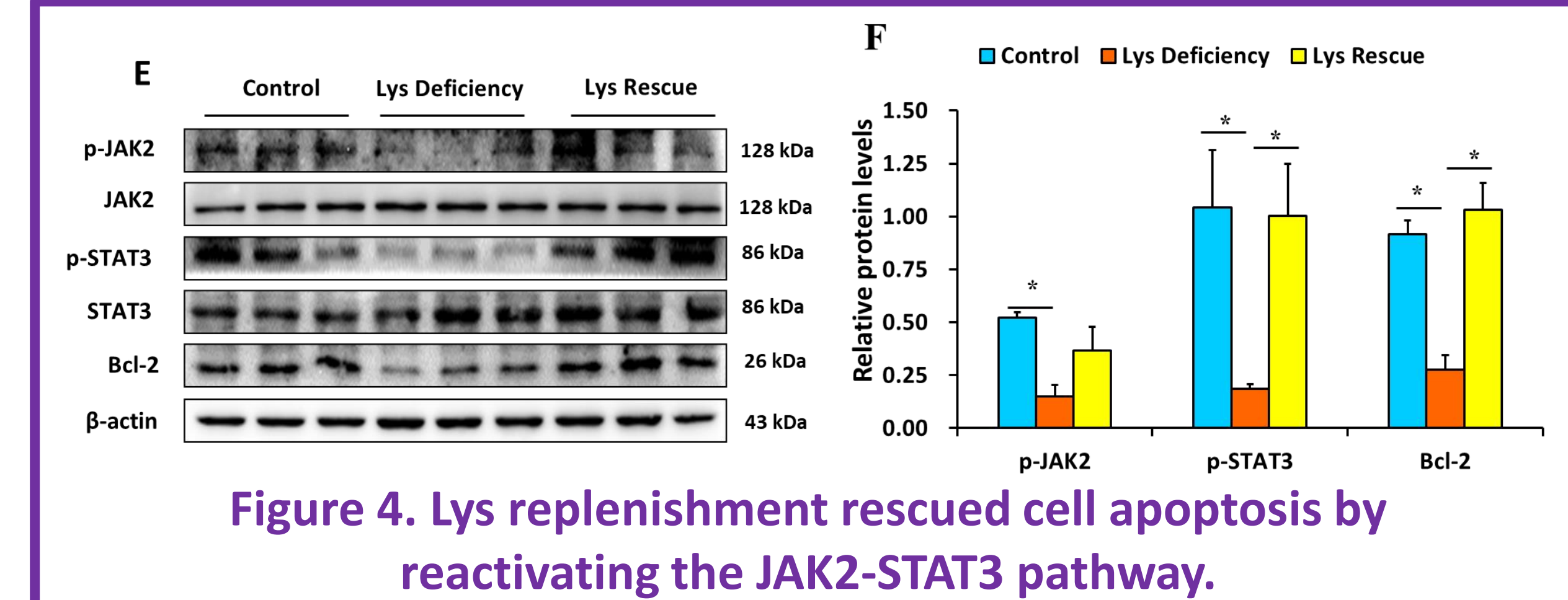
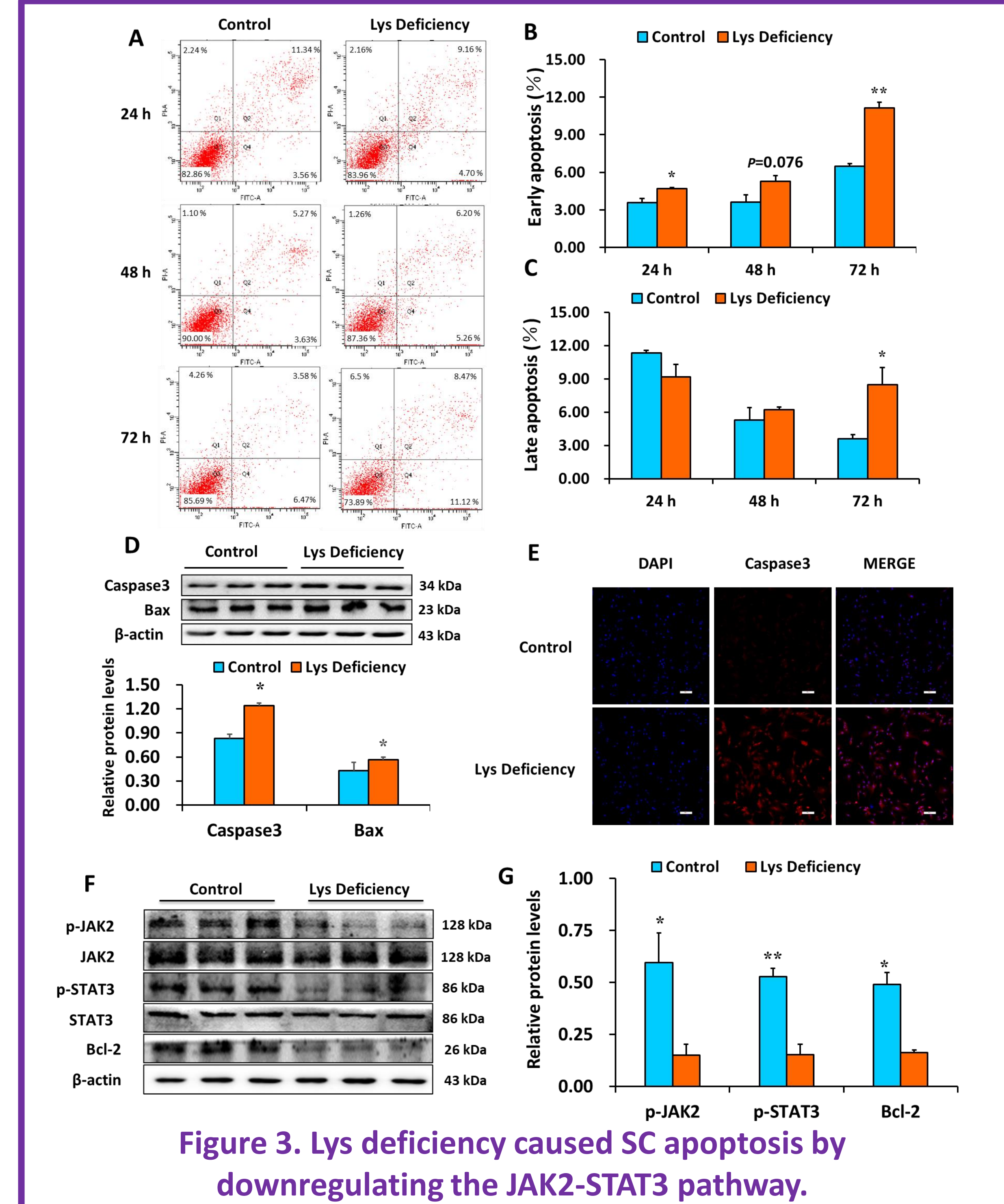
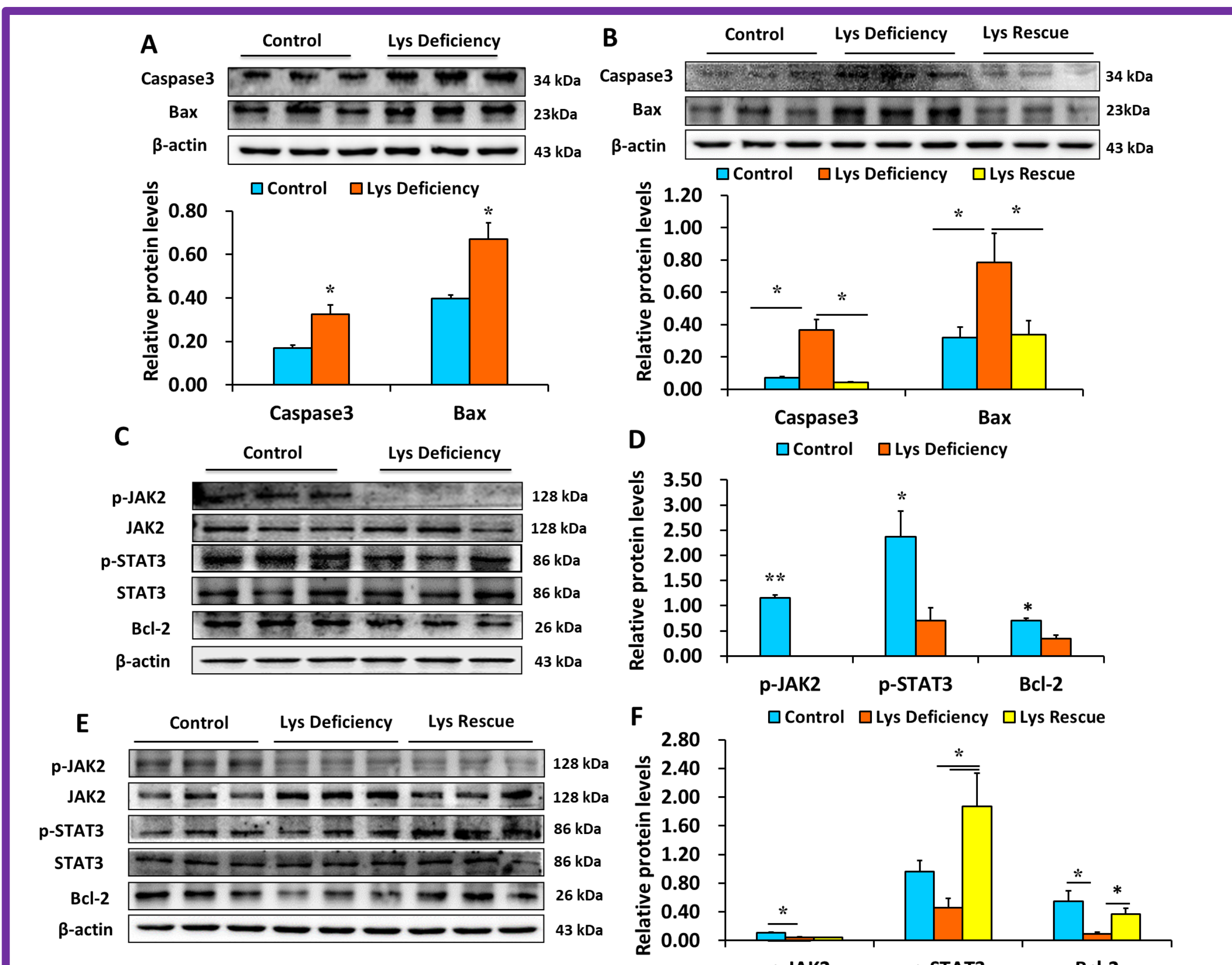
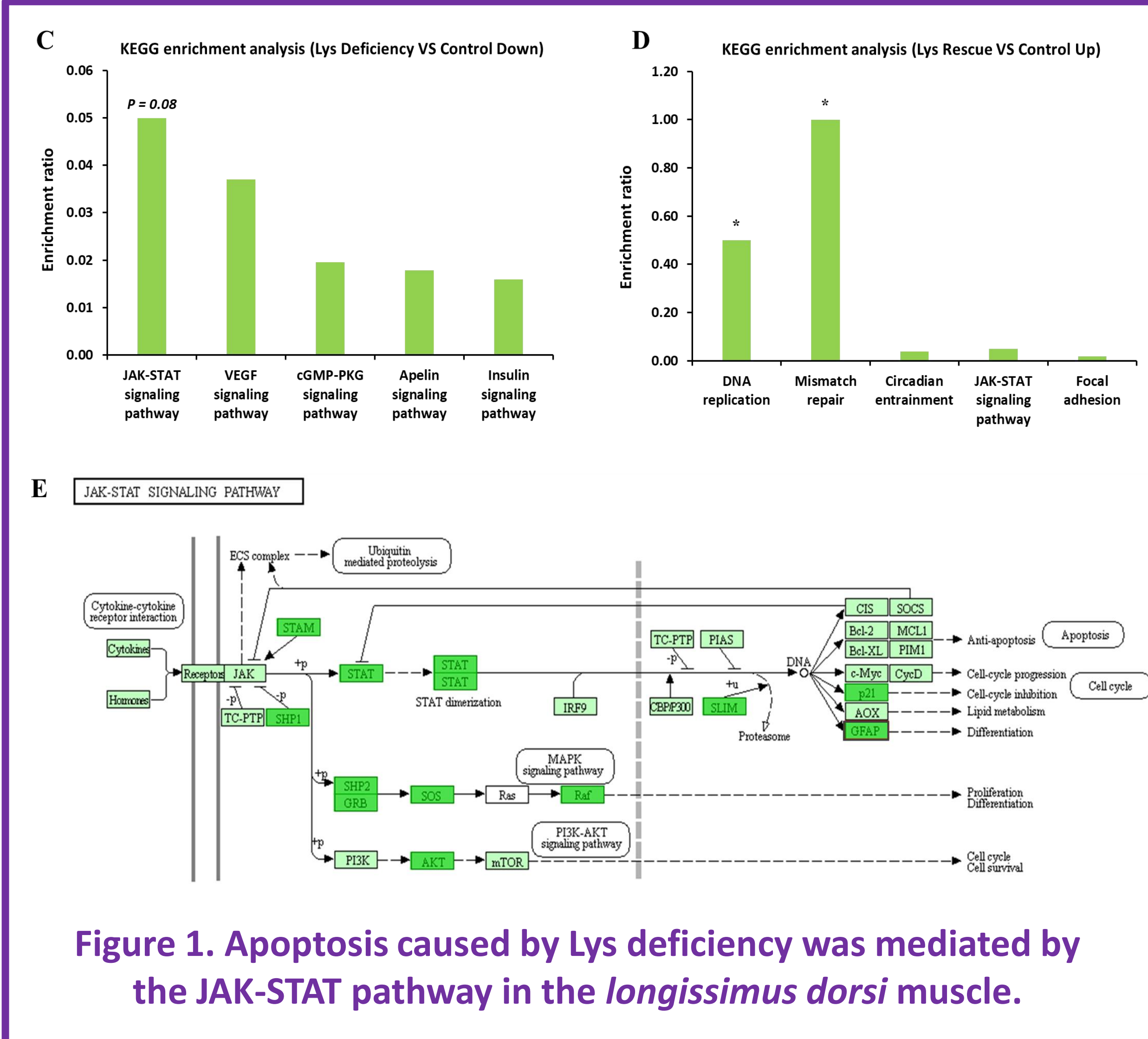
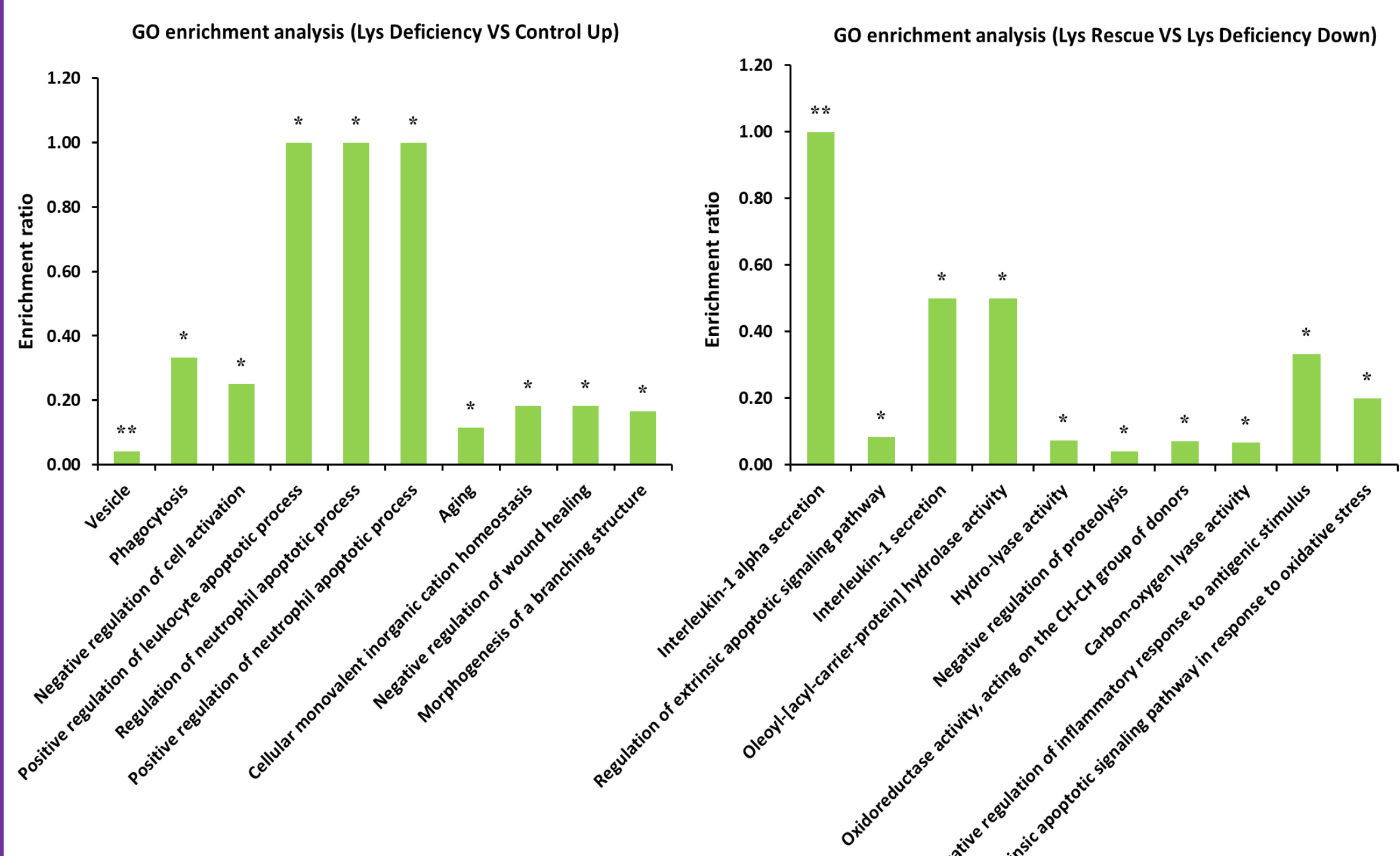
Experimental design



Materials and methods

- ◆ Crossbred weaned piglets (Duroc × Landrace × Large White)
- ◆ Skeletal muscle satellite cells (SCs)
- ◆ Lys depletion and replenishment
- ◆ JAK2 inhibitor assay
- ◆ iTRAQ-based quantitative proteomics analysis
- ◆ Flow cytometry
- ◆ Immunofluorescence staining
- ◆ Western blotting assay

Results



Conclusion
In conclusion, our findings demonstrate that the occurrence of porcine skeletal muscle SC apoptosis is dependent on Lys levels. Furthermore, Lys-dependent SC apoptosis is mediated by the JAK2-STAT3 pathway.

Acknowledgments
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Figure 2. Lys inhibited apoptosis in the longissimus dorsi muscle by upregulating the JAK2-STAT3 pathway.

Figure 3. Lys deficiency caused SC apoptosis by downregulating the JAK2-STAT3 pathway.

Figure 4. Lys replenishment rescued cell apoptosis by reactivating the JAK2-STAT3 pathway.

Figure 5. JAK2-STAT3 pathway inhibition caused cell apoptosis.