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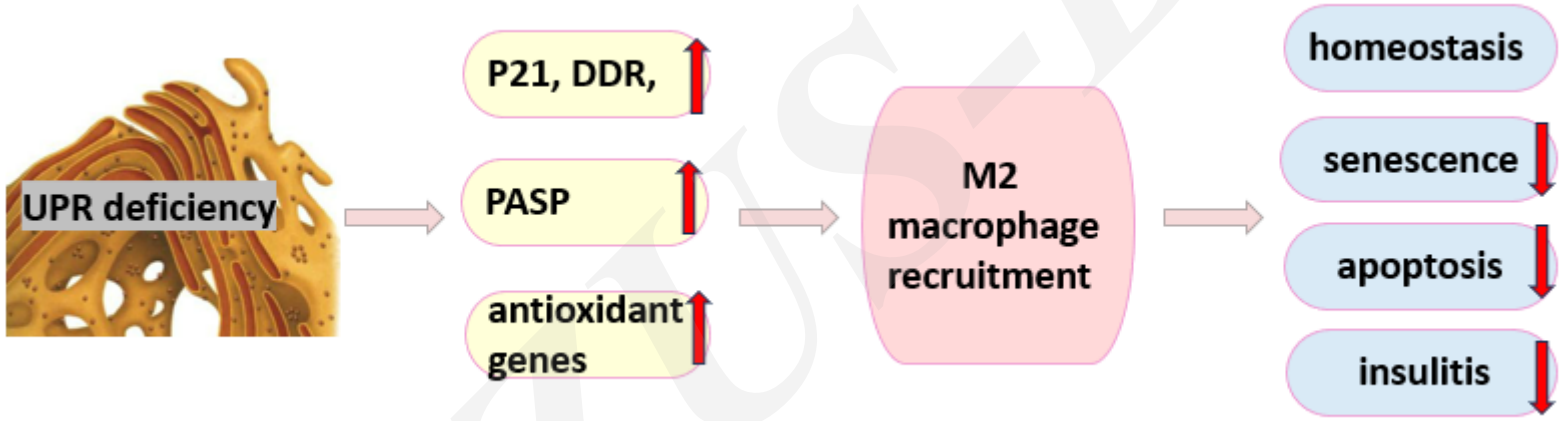
Early senescence of pancreatic β cells induced by unfolded protein response deficiency prevents type 1 diabetes

Key words: Type 1 diabetes (T1D), Senescence, Unfolded protein response (UPR), Pancreatic β, cells

Research Summary

This correspondence revealed an association between pancreatic β-cell UPR deficiency and cellular senescence, providing a novel preventive strategy for T1D.



- **UPR deficiency in NOD β cells triggers early senescence.**
- **p21-mediated secretome induces M2 macrophage recruitment to the islets.**
- **M2 macrophage promotes immune surveillance, eliminates terminal senesced β cells and reduces β cells apoptosis.**

Innovation points

- **Introduction** of various β-cell stresses, dysfunction, and death contributing to T1D pathogenesis.

- **Summary** of the most updated research progress about the role of β-cell ER stress in the development of T1D.

- **Emphasis** of the newly established association between β-cell UPR deficiency and cellular senescence.

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Figure 4