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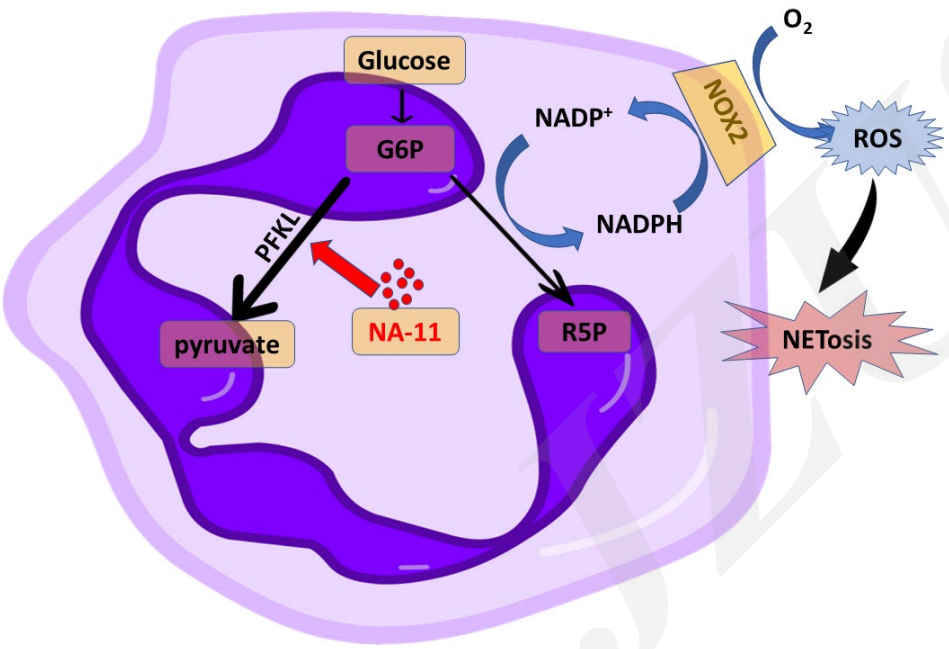
PFKL, a novel regulatory node for NOX2-dependent oxidative burst and NETosis

Key words: PFKL, NETosis, neutrophil, NOX

Research Summary

This perspective focused on a new strategy inhibiting aberrant NETosis to alleviate the severity of various inflammatory disorders.



- **NA-11 selectively activates PFKL to inhibit flux through the pentose phosphate pathway, thus limiting NADPH production.**
- **With the decrease of NADPH generation, NA-11 indirectly suppresses NOX2-dependent ROS production and NETosis, averting damage from aberrant NETosis.**
- **NA-11 maintains basal ROS generation, thus it maintains physiological NETosis to defend the host from infections.**

Innovation points

- **Introduction** of the inhibitors of completely suppressing G6PD or NOX2 activity to inhibit ROS production.

- **Summary** of the most updated research progress about inhibiting induced rather than basal activation of NOX2.

- **Emphasis** of a new small molecule, NA-11, activating PFKL to suppresses NETosis in neutrophils to avert damage from aberrant NETosis, but it still maintains physiological NETosis to defend the host from infections.

Figure 4