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Hypoxia potentiates LPS-induced inflammatory response and increases cell death by promoting NLRP3 inflammasome activation in pancreatic β cells

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Abstract

Hypoxia and islet inflammation are involved in β -cell failure in type 2 diabetes (T2D). Elevated plasma LPS levels have been verified in patients with T2D, and hypoxia occurs in islets of diabetic mice. Activation of inflammasomes in ischemic or hypoxic conditions was identified in various tissues. Here, we investigated whether hypoxia activates the inflammasome in β cells and the possible mechanisms involved. In mouse insulinoma cell line 6 (MIN6), hypoxia (1% O₂) primes the NLRP3 inflammasome along with NF- κ B signaling activation. Our results demonstrate that hypoxia can activate the NLRP3 inflammasome in LPS-primed MIN6 to result in initiating the β cell inflammatory response

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