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CONFÉRENCE

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PAVILLON ROGER GAUDRY
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Navigating the intricacies of the stress response and the innate immune response.

Endoplasmic reticulum (ER) stress triggers the integrated stress response (ISR) by activating PERK, which reduces global protein synthesis and helps cells adapt and survive. Recent studies reveal that STING can also activate PERK, leading to type I interferon production in response to viral DNA. Notably, specific ER stressors can stimulate PERK, enhancing STING and interferon production, especially in human plasmacytoid dendritic cells. A case study of a patient with STING-associated vasculopathy highlights the critical role of PERK-STING interaction in innate immunity. The patient's PERK mutation impairs monocyte differentiation and amplifies STING signaling, boosting inflammatory mediators. This variant also activates the NRF2/HO-1 and NF-κB pathways. While cytokine mRNA expression increases, protein translation is controlled, revealing a novel level of regulation. These findings offer insights into the ISR's role in monocyte differentiation and STING activation, impacting interferonopathies and autoimmune diseases.

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