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Gates of inflammation on the mitochondrial membrane

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Mitochondrial stress releases mitochondrial DNA (mtDNA) into the cytosol, triggering immunostimulatory pathways such as the type-I interferon response. Pores formed in the outer membrane of mitochondria (OMM) can promote mtDNA release; however, the identity of such a pore in living cells is not well characterized. Here, we provide genetic and biochemical evidence demonstrating that the oligomerization of voltage-dependent anion channel 1 (VDAC1) in the OMM promotes mtDNA release and triggers the type-I interferon response in living cells. In addition to forming OMM pores, VDAC1 interacts with mtDNA via its positively charged residues in the N-terminal domain and increases both VDAC1 oligomerization and type-I interferon response. VBIT-4, which inhibits VDAC1 oligomerization, decreases mtDNA release, type-I interferon signaling, neutrophil extracellular traps, and disease severity in a mouse model of systemic lupus erythematosus. Thus, inhibiting VDAC oligomerization is a potential therapeutic approach for diseases associated with mtDNA release.

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