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OPA1 promotes pH flashes that spread between contiguous mitochondria without matrix protein exchange

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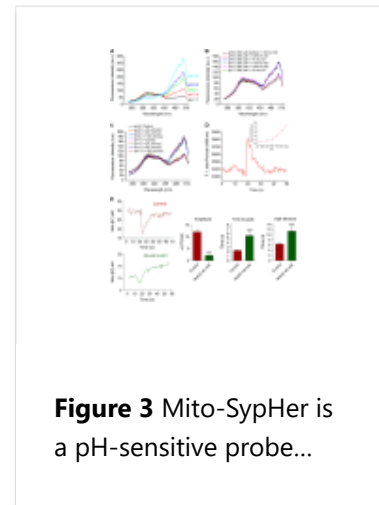
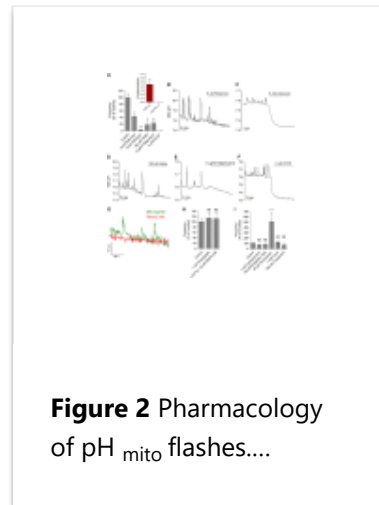
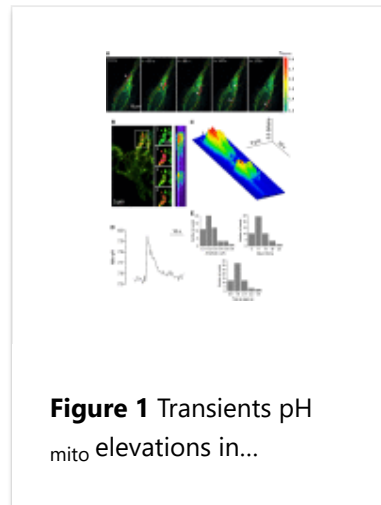
Abstract

The chemical nature and functional significance of mitochondrial flashes associated with fluctuations in mitochondrial membrane potential is unclear. Using a ratiometric pH probe insensitive to superoxide, we show that flashes reflect matrix alkalinization transients of ~0.4 pH units that persist in cells permeabilized in ion-free solutions and can be evoked by imposed mitochondrial depolarization. Ablation of the pro-fusion protein Optic atrophy 1 specifically abrogated pH flashes and reduced the propagation of matrix photoactivated GFP (paGFP). Ablation or invalidation of the pro-fission Dynamin-related protein 1 greatly enhanced flash propagation between contiguous mitochondria but

marginally increased paGFP matrix diffusion, indicating that flashes propagate without matrix content exchange. The pH flashes were associated with synchronous depolarization and hyperpolarization events that promoted the membrane potential equilibration of juxtaposed mitochondria. We propose that flashes are energy conservation events triggered by the opening of a fusion pore between two contiguous mitochondria of different membrane potentials, propagating without matrix fusion to equilibrate the energetic state of connected mitochondria.

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Figures



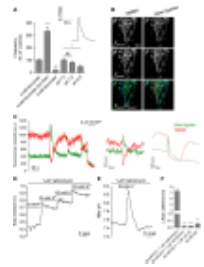


Figure 4 pH_{mito} flashes are bioenergetic...

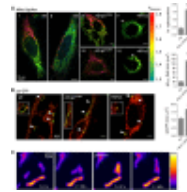


Figure 5 Matrix pH elevations propagate along...

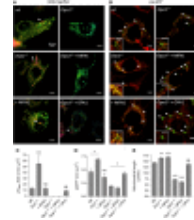


Figure 6 pH_{mito} flashes require OPA1...

All figures (8)

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