



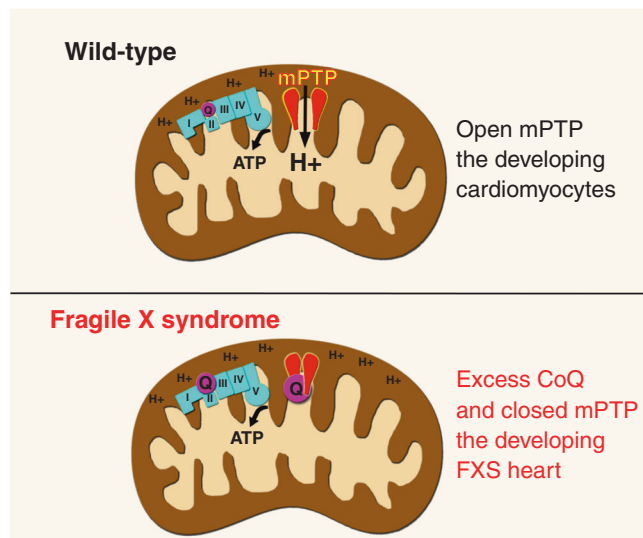
IMAGE

Insights image for “The newborn *Fmr1* knockout mouse: a novel model of excess ubiquinone and closed mitochondrial permeability transition pore in the developing heart”

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Pediatric Research (2021) 89:707; <https://doi.org/10.1038/s41390-020-01144-4>

The mitochondrial permeability transition pore (mPTP) is a source of proton leak and is physiologically open in the developing wild-type heart. Newborn Fragile X syndrome (FXS) cardiomyocyte mitochondria have excess coenzyme Q (CoQ), less proton leak, and a closed mPTP. CoQ is likely an important regulator of the mPTP during development.



ACKNOWLEDGEMENTS

This work was supported by NIH/NINDS R01NS112706 (to R.J.L.).

ADDITIONAL INFORMATION

Competing interests: The authors declare no competing interests.

Patient consent: Patient consent was not required.

Publisher's note Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

REFERENCE

Barajas, M. et al. The newborn *Fmr1* knockout mouse: a novel model of excess ubiquinone and closed mitochondrial permeability transition pore in the developing heart. *Pediatr. Res.* <https://doi.org/10.1038/s41390-020-1064-6> (2020).

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Received: 11 August 2020 Accepted: 11 August 2020

Published online: 12 September 2020