JUL 31, 2021

OPEN ACCESS

DOI:
dx.doi.org/10.17504/protocols.io.bswanfae

Collection Citation: Odetta Antico, Miratul M. Muqit 2021. Cell-based analysis of PINK1-Parkin pathway activation in primary mouse cortical neurons. **protocols.io**
<https://dx.doi.org/10.17504/protocols.io.bswanfae>

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Protocol status: Working
 We use this protocol and it's working

Created: Mar 01, 2021

Last Modified: Dec 26, 2022

COLLECTION integer ID:
 47778

Keywords: Neurons, PINK1, Parkin, Mitochondrial stress, ubiquitin, ASAPCRN

🌐 Cell-based analysis of PINK1-Parkin pathway activation in primary mouse cortical neurons

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ABSTRACT

Mutations in PINK1 cause early-onset Parkinson's disease. PINK1 becomes stabilised and active upon mitochondrial depolarisation. This leads to phosphorylation of ubiquitin and Parkin via Serine 65 residues and a feed forward mechanism whereby PINK1 phosphorylates newly formed polyubiquitin chains, generating phospho-ubiquitin, which further promotes Parkin recruitment and activation. Once activated, Parkin ubiquitylates proteins at the outer face of the outer mitochondrial membrane (OMM) and then initiates a downstream pathway that eventually leads to mitophagy, a mitochondria-specific type of autophagy. Notably, much of previous investigation into PINK1/Parkin activity has been performed in non-neuronal human cancer cells where Parkin and/or PINK1 is over-expressed. Here we report a protocol for generation of mouse embryonic cortical neuronal cultures that produce high cell yields and can be used for studying endogenous PINK1 and Parkin signalling by biochemical methods and proteomics.

ATTACHMENTS

[Mouse PINK1 pathway protocol \(166 - 337\).pdf](#)

ATTACHMENTS

Mouse PINK1
pathway
protocol (166
- 337).pdf

FILES

Protocol

NAME
PROCEDURE TO INDUCE MITOCHONDRIAL DEPOLARISATION AND LYSING OF MOUSE CORTICAL NEURONS

VERSION 1

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Protocol

NAME
PROCEDURE TO ISOLATE AND CULTURE NEURONS FROM EMBRYONIC MOUSE CORTEX

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