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Lab Resource: Genetically-Modified Single Cell Line

CRISPR/Cpf1-mediated editing of PINK1 in induced pluripotent stem cells

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A B S T R A C T

The PTEN induced kinase 1 (*PINK1*) gene is crucial for mitophagy and mitochondrial quality control. Mutations in the *PINK1* gene are associated with several neurological disorders. To decipher the role of PINK1-mediated mitophagy in human induced pluripotent stem cells (hiPSCs) and in their differentiated counterparts, we used CRISPR/Cpf1 and generated a human iPSC line with homozygous out-of-frame deletions by targeting exon 6 of the *PINK1* gene. The generated homozygous *PINK1* mutant cell line showed normal cell morphology, genomic stability, and expression of classical stem cell markers. Furthermore, the cells can be differentiated efficiently into the three germ layers.

Resource Table: Please fill in the right-hand column of the table below. All information requested in the table is MANDATORY where indicated. Manuscripts with incomplete or incorrect information will be sent back for completion. Many entries apply to the PARENTAL line, the relationship to which has to be demonstrated via the establishment of the underlying pairwise genomic equivalence (e.g. STR analysis).

Unique stem cell line identifier	IUFi021-A-10
Alternative name(s) of stem cell line	DU466
Institution	IUF Leibniz research institute for Environmental Medicine, Heinrich-Heine Universität Düsseldorf (HHU)
Contact information of the reported cell line distributor	Andrea Rossi; andrea.rossi@iuf-duesseldorf.de Andreas Reichert; reichert@hhu.de
Type of cell line	Induced pluripotent stem cells (iPSC)
Origin	Human
Additional origin info *(applicable for human ESC or iPSC)*	Age: fetal Sex: female
Cell Source	IPS(IMR90)-4 (WISCi004-B)
Method of reprogramming	Lentiviral transduction of *POU5F1*, *SOX2*, *NANOG*, *LIN28*
Clonality	Monoclonal

(*continued on next column*)

(*continued*)

Unique stem cell line identifier	IUFi021-A-10
Evidence of the reprogramming transgene loss (including genomic copy if applicable)	Not applicable
The cell culture system used	Geltrex coated plates and mTeSR + as growth media
Type of the Genetic Modification	Induced Mutation (out-of-frame indels)
Associated disease	
Gene/locus modified in the reported transgenic line	PTEN induced kinase 1 (*PINK1*)
Method of modification / user-customisable nucleases (UCN) used, the resource used for design optimisation	CRISPR/Cas12a (Cpf1)
User-customisable nuclease (UCN) delivery method	Ribonucleoprotein (RNP)
All double-stranded DNA genetic material molecules introduced into the cells	No double stranded DNA molecules introduced
Evidence of the absence of random integration of any plasmids or DS DNA introduced into the cells.	Not applicable
Analysis of the nuclease-targeted allele status	Next generation sequencing of targeted allele (MiSeq)

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(*continued*)

Unique stem cell line identifier	IUFi021-A-10
Homozygous allele status validation	Next generation sequencing of targeted allele (MiSeq)
Method of the off-target nuclease activity prediction and surveillance	No off-targets were predicted for guide RNAs by CHOP-CHOP even after three mismatches
Descriptive name of the transgene	No transgene cassette used
Eukaryotic selective agent resistance cassettes (including inducible, gene/cell type-specific)	No selection marker used for selecting transfected cells
Inducible/constitutive expression system details	No inducible or Constitutive expression
Date archived/stock creation date	06.08.2025
Cell line repository/bank	Registered in the hpscreg database. Biosample IDs: SAMEA118839125
Ethical/GMO work approvals	Commercially available human female IPS(IMR90)-4 (WISCi004-B) derived from reprogrammed fetal lung fibroblasts were purchased from the WiCell Company and are available for Research Use Only (RUO).
Addgene/public access repository recombinant DNA sources' disclaimers (if applicable)	Not applicable

The manuscript section expected contents clarification

1. Resource utility

The *PINK1* mutant iPSC line generated in this study provides a relevant model system and valuable resource to investigate the role of mitochondrial quality control and PINK1-mediated mitophagy in induced pluripotent stem cells (iPSCs) and in iPSC-derived differentiated cells including cardiomyocytes (iPSC-CMs) Table 1.

2. Resource details

Mitochondria are dynamic organelles enclosed by two membranes executing multiple vital functions in eukaryotic cells (Kondadi and Reichert, 2024). Mitochondria serve as a center for energy metabolism producing most of the chemical energy resource in the form of adenosine triphosphate (ATP) via oxidative phosphorylation. Beyond energy conversion, mitochondria also play crucial roles in cell growth and differentiation, cell signaling, regulation of programmed cell death, immunity, cell cycle control, fatty acid oxidation, heme synthesis, intracellular calcium homeostasis, and iron-sulfur biogenesis (Malek et al., 2018; Reichert and Neupert, 2004). Mitochondria are able to interconvert in a dynamic fashion from a highly interconnected tubular network by membrane fusion to fragmented or clustered organelles by fission, and vice versa (Kondadi and Reichert, 2024; Rossmann et al., 2021). Mitochondrial homeostasis and quality control is required for maintaining several proper cellular functions. Mitophagy, the selective engulfment of mitochondria by autophagosomes, followed by their transport to and elimination by lysosomes, is one of the important mitochondrial quality control pathways, maintaining mitochondrial homeostasis (Rossmann et al., 2021). The PTEN induced kinase 1 (*PINK1*) plays an important role in mitophagy as it phosphorylates ubiquitin and PARKIN, an E3 ubiquitin ligase, required to activate this pathway (Narendra et al., 2010). Several roles of mitophagy have been identified in differentiation process and heart development. Therefore, mutation or genetic deletion of *PINK1* affects cellular energy, reactive oxygen species (ROS) production, intracellular calcium levels, and the production of apoptotic signals, causing numerous cardiovascular diseases and Parkinson's disease (Cho et al., 2013; Rossmann et al., 2021).

Table 1
Characterization and validation.

Classification (Optional *italicized*)	Output type	Result	Data
Schematic of a transgene/genetic modification			
Morphology	Photography Bright field	A typical iPSC morphology	Fig. 1C
Pluripotency status evidence for the described cell line	hiPSCore analysis qPCR: Gene expression and staining of OCT-4 and SOX-2 (undifferentiated markers)	Cell lines passed hiPSCore analysis for pluripotency and positive to OCT-4 and SOX-2 IF markers	Fig. 1D, E
Karyotype	Karyotype (G-banding)	46XX	Fig. 1F
Genotyping for the desired genomic alteration/allelic status of the gene of interest	PCR across the edited site or targeted allele-specific PCR	Next generation sequencing for the targeted region	Fig. 1B
	Evaluation of the – (homo-/hetero-/hemi-) zygous status of introduced genomic alteration(s)	Not applicable	Not applicable
	Transgene-specific PCR (when applicable)	Not applicable	Not applicable
Verification of the absence of random plasmid integration events	Not applicable	Not applicable	Not applicable
Parental and modified cell line genetic identity evidence	CNV analysis		
Mutagenesis / genetic modification outcome analysis	Next generation sequencing of the targeted regions by MiSeq analysis	Confirmation of the precise nature of introduced alteration, also heterozygous/homozygous state of mutation(s)	Fig. 1B
Off-target nuclease activity analysis	No indication of off-targets even allowing more than three mismatches	Not applicable	Not applicable
Specific pathogen-free status	Mycoplasma	Standard PCR analysis	Fig. 1G
Multilineage differentiation potential	In- vitro differentiation into germ layers ectoderm, endoderm and mesoderm followed by hiPSCore analysis qPCR: Gene expression	Cell lines can be differentiated into three germ layers and passed hiPSCore analysis	Fig. 1E
List of recommended germ layer markers	In- vitro differentiation into germ layers ectoderm, endoderm and mesoderm followed by hiPSCore analysis qPCR: Gene expression & IF microscopy	Pluripotent: *CNMD, NANOG, SPP1* & IF with *OCT-4* and *SOX-2* antibodies Ectoderm: *HES5, PAMR1, PAX6* Endoderm: *CER1, EOMES, GATA6* Mesoderm:	RT-PCR or qRT-PCR with reference gene(s) and IF with specific antibodies

(*continued on next page*)

Table 1 (*continued*)

Classification (Optional *italicized*)	Output type	Result	Data
		APLNR, HAND1, HOXB7	
Outcomes of gene editing experiment (OPTIONAL)	Brief description of the outcomes in terms of clones generated/ establishment approach/screening outcomes	Not tested	–
Donor screening (OPTIONAL)	HIV 1 + 2 Hepatitis B, Hepatitis C	Not tested	–
Genotype – additional histocompatibility info (OPTIONAL)	Blood group genotyping	Not tested	–
	HLA tissue typing	Not tested	–

IMR90 cell line with *PINK1* mutation was generated using CRISPR/Cas12a (Cpf1) based approach as shown in (Fig. 1A). Briefly, iPS cells were electroporated with Cpf1 guide RNA and protein. Single clones were generated and genotyped by next generation sequencing (Ramachandran et al., 2021) to identify successful edits (out-of-frame mutations within Exon 6 of the *PINK1* gene) (Fig. 1B). One of the successfully edited clone was then expanded for further analysis. The selected clone exhibits typical iPSC morphology characterized by compact colonies, a high nucleus-to-cytoplasm ratio, and well-defined borders (Fig. 1C). Furthermore, the selected clone was stained for pluripotency markers OCT-4 and SOX-2, confirming the heterogeneity of the cell population in the undifferentiated state of these edited clones (Fig. 1D). The differentiation potential was validated using hiPSCore analysis (Dobner et al., 2024), confirming the pluripotent state of the cells (Fig. 1E). G-banding analysis confirms normal karyotypes, indicating chromosomal integrity post-editing (Fig. 1F). Copy number variation (CNV) analysis further ensures the absence of chromosomal aberrations and also high genomic concordance between the edited clones and the parental line (Supplementary Fig. 1). The cells were tested negative for mycoplasma using standard PCR based assay (Fig. 1G). No off-target sites were detected for the selected guide RNA, even when allowing more than three mismatches.

3. Materials and methods

3.1. Cell culture

Human female IMR90 iPS cells (WISCi004-B) were cultured in mTeSR™ Plus medium (STEMCELL Technologies) with 5X supplement and 1 % Penicillin-Streptomycin (complete medium) on 1 % Geltrex™-coated (Gibco) plates at 37 °C and 5 % CO_2. For maintenance, cells were passaged using enzyme-free ReLeSR™ (STEMCELL Technologies). To generate single cell suspensions, cells were spilitted with Accutase (Pan Biotech) and seeded in complete medium with 10 μM Y-27632 (Selleckchem) or with CEPT (R&D Systems). The cells were kept for a maximum of 10 passages after thawing.

3.2. Generation and genotyping of IPS(IMR90)-4-derived homozygous PINK1 mutants

Guide RNAs were designed using CHOPCHOP (https://chopchop.cbu.uib.no/) (Labun et al., 2019). Guide RNA sequences are listed in Table 2. Wild type IMR90 cells were electroporated with RNPs using the NEON electroporation system. Electroporation conditions were 1400 Voltage (V) for 20 ms (ms). Single clones were generated and genotyped by deep sequencing using a MiSeq (Illumina) bench-top system as described previously (Ramachandran et al., 2021) using a Nano V2

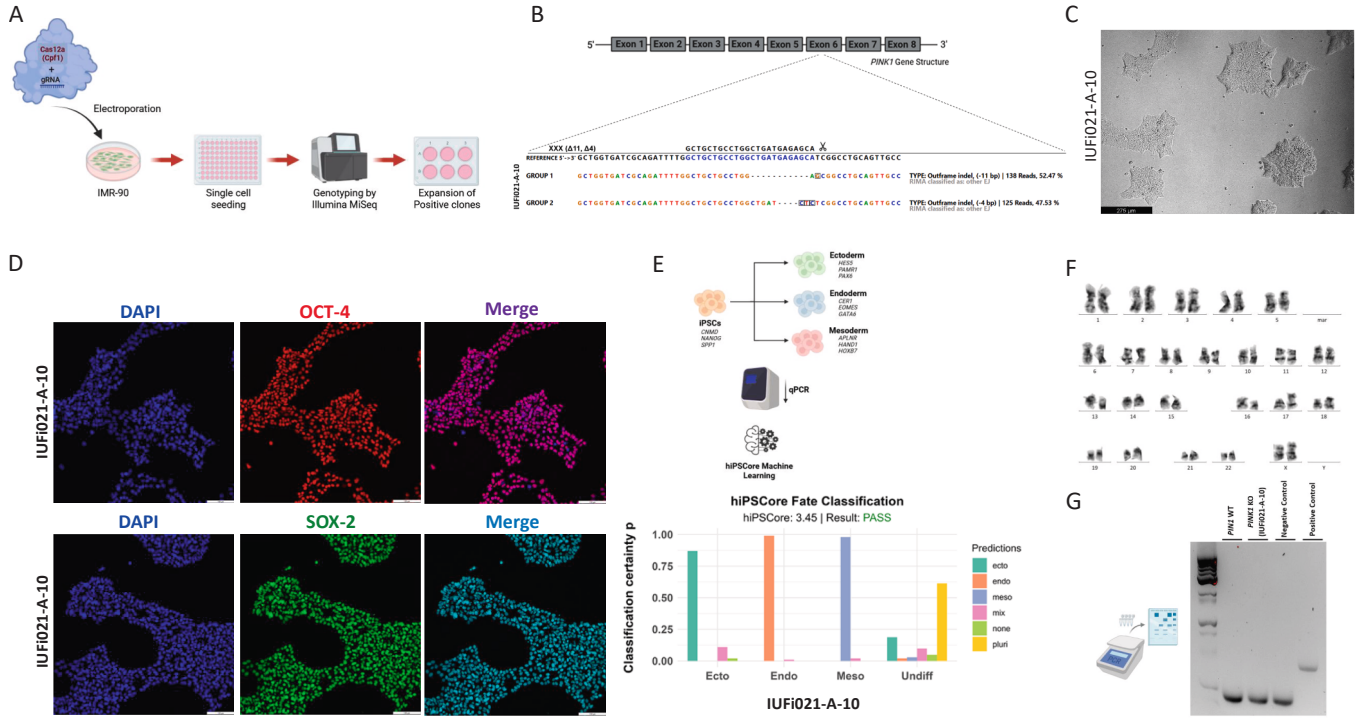

Fig. 1. Characterization of the CRISPR/Cpf1-engineered PINK1 mutant iPSC line (IUFi021-A-10). **A,** Strategy for introducing PINK1 mutation into IMR90 iPSCs using a CRISPR/Cas12a (Cpf1)–based gene-editing strategy. **B,** Single-cell–derived clones were expanded and genotyped by next-generation DNA sequencing revealing out-of-frame mutations within exon 6 of the PINK1 gene. **C,** selected mutant clone exhibited typical iPSC colony morphology including compact growth, a high nucleus-to-cytoplasm ratio, and defined colony borders. **D,** immunostaining for OCT-4 and SOX-2 confirmed expression of pluripotency markers in selected clone. **E,** hiPSCore analysis demonstrating retention of trilineage differentiation potential and validated pluripotency. **F,** G-banding karyotype analysis indicating normal chromosomal integrity following genome editing. **G,** PCR-based confirmation of absence of contamination by *Mycoplasma* species.

Table 2
Reagents details. *RRID Requirement for antibodies: use http://antibodyregistry.org/ to retrieve RRID for antibodies and include ID in the table as shown in examples.*

Antibodies and stains used for immunocytochemistry/flow-cytometry			
	Antibody	Dilution	Company Cat # and RRID
Pluripotency Markers	Oct-4A (C30A3) Rabbit mAb	1:400	Cell Signalling 2840 RRID: AB_2167691
Pluripotency Markers	Sox2 (D6D9) XP Rabbit mAb	1:400	Cell Signalling 67,789 RRID: AB_2799734
Secondary antibodies	Goat anti-Rabbit IgG (H + L) Highly Cross-Adsorbed Secondary Antibody, Alexa Fluor 488	1:500	(Thermo Fisher Scientific A32731TR) RRID: AB_2866491
Secondary antibodies	Goat anti-Rabbit IgG (H + L) Highly Cross-Adsorbed Secondary Antibody, Alexa Fluor™ Plus 555	1:500	(Thermo Fisher Scientific A32732) RRID: AB 266281
e.g. Nuclear stain	Hoechst 33,342	1 μg/mL	Thermoscientific R37165 RRID: 2651133
Site-specific nuclease			
Nuclease information	Nuclease type/version	Alt-R™ CRISPR-Cas12a (Cpf1)	
Delivery method	Electroporation (parameters)	Neon/Invitrogen-TFS, 1x (1400 V, 20 ms)	
Selection/enrichment strategy	Selection cassette(s), FACS	Not applicable	
Primers and Oligonucleotides used in this study			
	Target	**Forward/Reverse primer (5′-3′)**	
Genotyping (desired allele/transgene presence detection)	*PINK1* Illumina MiSeq	ACACTCTTTCCCTACACGACGctcttccgatctTGGTGGCTTTAGTAGGGACATAG / TGACTGGAGTTCAGACGTGTGctcttccgatctATTCAGTGGACATGTGGGGGAAG	
Targeted mutation analysis/sequencing	Sequencing data from both alleles	Miseq output files	
Potential random integration-detecting PCRs	Not-applicable	Not-applicable	
Cas12 (Cpf1) gRNA	*PINK1*	GCTGCTGCCTGGCTGATGAGAGCA	
Genomic target sequence(s)	Including PAM and other sequences likely to affect UCN activity	Including exact position in the reference genome (e.g. GRCh38 for human cell lines)	
hiPSCore primers	Lineage-specific markers as described in (Dobner et al., 2024). See below:	–	
House-Keeping Genes (qPCR)	*ACTB*	TGAGGCACTCTTCCAGCCTTC/CGGCAATGCCAGGGTACATG	
House-Keeping Genes (qPCR)	*GAPDH*	GTCTCCTCTGACTTCAACAGCG/ACCACCCTGTTGCTGTAGCCAA	
Mesoderm marker (qPCR)	*APLNR*	TTGCAGAGTGGGTGACAGAG/CTGGTTGTCTGCCCCATAGT	
Mesoderm marker (qPCR)	*HAND1*	ACATCGCCTACCTGATGGAC/CGGCTCACTGGTTTAACTCC	
Mesoderm marker (qPCR)	*HOXB7*	ATCTACCCCTGGATGCGAAGCT/GCGTCAGGTAGCGATTGTAGTG	
Endoderm marker (qPCR)	*CER1*	CCCATCAAAAGCCATGAAGT/AATGAACAGACCCGCATTTC	
Endoderm marker (qPCR)	*GATA6*	TGTGCGTTCATGGAGAAGATCA/TTTGATAAGAGACCTCATGAACCGACT	
Endoderm marker (qPCR)	*EOMES*	GGCTGTCTCCTAGCAACTCC/GCATAATACCCTCCCATGCCT	
Ectoderm marker (qPCR)	*HES5*	CTGCTCAGCCCCAAAGAG/GCTCGATGCTGCTGTTGAT	
Ectoderm marker (qPCR)	*PAMR1*	TTGCCAGCAGAATGGAGAGTGG/CTTGACTGAACCTGCATCGGAAG	
Ectoderm marker (qPCR)	*PAX6*	GAGGTCAGGCTTCGCTAATG/TTGCTTGAAGACCACAATGG	
Pluripotency marker (qPCR)	*CNMD*	CCGTGACCAAACAGAGCATCTC/CTGTTGTCCTTCACAGGCTGATC	
Pluripotency marker (qPCR)	*SPP1*	CGAGGTGATAGTGTGGTTTATGG/GCACCATTCAACTCCTCGCTTTC	
Pluripotency marker (qPCR)	*NANOG*	TCCAACATCCTGAACCTCAG/ACCATTGCTATTCTTCGGCC	

Cassette. Primer details are given in Table 2. FASTQ files were analyzed using Edit-o-Matic (Nguyen, 2025) and CleanFinder (Spiessbach et al., 2025).

3.3. Karyotyping

Karyotyping analysis was performed at the Institute of Human Genetics and Anthropology of the Heinrich-Heine-University, Düsseldorf.

3.4. Mycoplasma test

Mycoplasma contamination was assessed using the Mycoplasma Detection Kit (SouthernBiotech) according to the manufacturer's instructions.

3.5. Analysis of submicroscopic chromosomal variations and copy number variations (CNVs)

Low resolution karyotyping was conducted on an Illumina Infinium Global Screening GSAMD-24v3-0-EA_20034606 Array by Life&Brain. Data analysis was conducted with GenomeStudio V2.0. CNV and LOH analysis was performed via the CNV Partition 3.2 Plugin algorithm. CNVs encompassing less than 15 SNPs and regions smaller than 30 kb were not considered.

3.6. hiPSCore analysis

Functional pluripotency analysis of the IUFi021-A-10 cell line was performed using the machine learning–based hiPSCore classification system, as previously described (Dobner et al., 2024). Briefly, IUFi021-A-10 cells were differentiated into ectoderm, mesoderm, and endoderm lineages using the STEMdiff Trilineage Differentiation Kit (Stemcell Technologies), following the manufacturer's instructions. RNA was isolated from each differentiated lineage as well as from undifferentiated cells. Quantitative PCR was conducted using primers for pluripotency and lineage-specific markers, and the results were evaluated with the hiPSCore scoring algorithm. This machine learning–based system integrates data from a wide range of commercially available and genetically modified human iPSC lines, including KOL2.1J, iPS12, and IMR90, enabling robust and comparative assessment of functional pluripotency (Fig. 1E).

3.7. Immunofluorescent imaging

Selected clone (iPSCs) was fixed in 4 % PFA for 15 min and permeabilized with 0.1 % Triton X-100 for 1 h at RT. Fixates were incubated with 3 % BSA for 2 h and incubated overnight with respective primary antibodies at 4 °C. Fixates were incubated with Alexa-Fluor-conjugated secondary antibodies for 2 h at RT, and nuclei were stained with Hoechst 33,258 (Sigma-Aldrich). Antibodies are listed in Table 2. Images were obtained using Leica DMi8 (Leica) fluorescence microscope.

Ethics

Commercially available human female IPS(IMR90)-4 lines (WISCi004-B) derived from reprogrammed fetal lung fibroblasts were purchased from the WiCell Company and are available for Research Use Only (RUO).

CRediT authorship contribution statement

Roohallah Ghodrat: Data curation, Formal analysis, Investigation, Methodology, Visualization, Writing – original draft, Writing – review & editing. **Haribaskar Ramachandran:** Data curation, Formal analysis, Investigation, Methodology. **Barbara Hildebrandt:** Data curation, Formal analysis, Methodology. **Stephanie Binder:** Data curation, Formal analysis, Methodology. **Andrea Rossi:** Conceptualization, Funding acquisition, Supervision, Writing – review & editing. **Andreas S. Reichert:** Conceptualization, Funding acquisition, Project administration, Supervision, Writing – original draft, Writing – review & editing.

Declaration of competing interest

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests: Andreas S. Reichert reports financial support was provided by German Research Foundation. Andrea Rossi reports financial support was provided by German Research Foundation. Andrea Rossi reports financial support was provided by VHL von Hippel-Lindau Betroffene Familien e. V. Andrea Rossi reports financial support was provided by AFM-Téléthon. Andrea Rossi reports financial support was provided by European Union and North Rhine-Westphalia (NRW) Start-up grant. Andrea Rossi reports financial support was provided by Leibniz Competition (SAW) Cooperative Excellence project. Andrea Rossi serves on the editorial board of Stem Cell Research (SCR). If there are other authors, they declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.scr.2025.103887.

Data availability

Data will be made available on request.

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