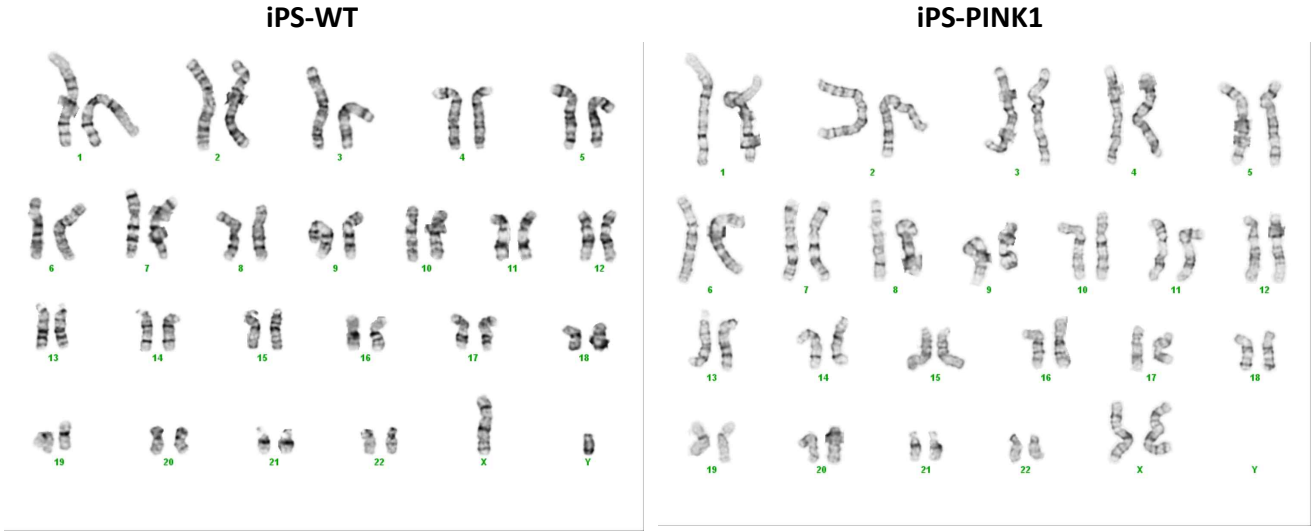

Fig. S1. Karyotype analysis of generated iPS cell lines iPS-WT and iPS-PINK1. Both iPS cell lines displayed a normal karyotype.

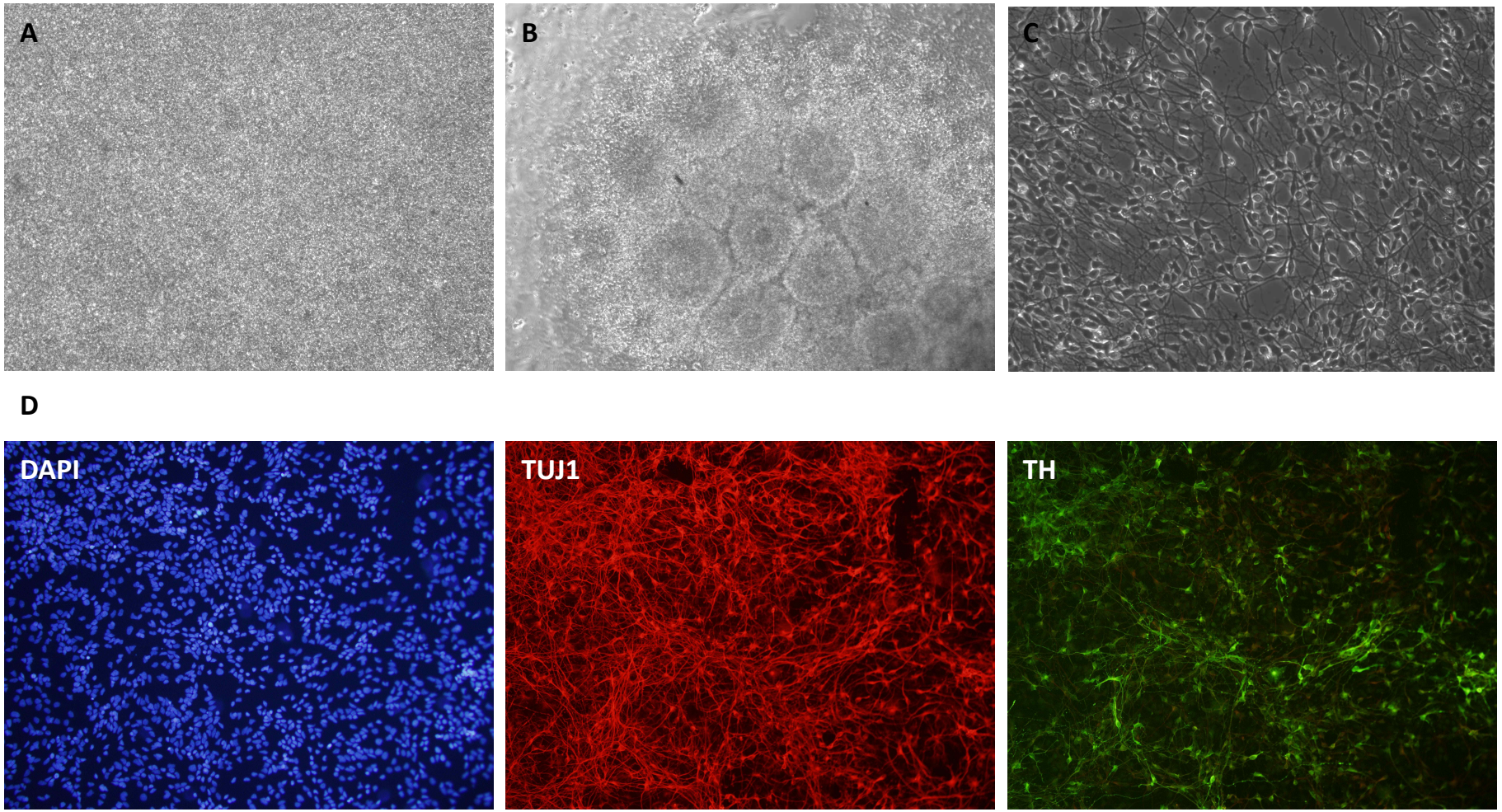

Fig. S2. Stages during neuronal differentiation. **(A)** On day 11 of differentiation, cells have formed a dense layer. After passaging *en bloc*, neural rosette structures were seen on day 16 **(B)**, indicating the presence of premature CNS cells. **(C)** On day 30 of differentiation, neurogenesis was largely completed with a large proportion of neurons growing in a monolayer.
(D) Images that were used for cell counts. Neurons were co-immunostained with nuclear marker DAPI (total cells), neuronal marker TUJ1 and dopaminergic marker tyrosine hydroxylase (TH).

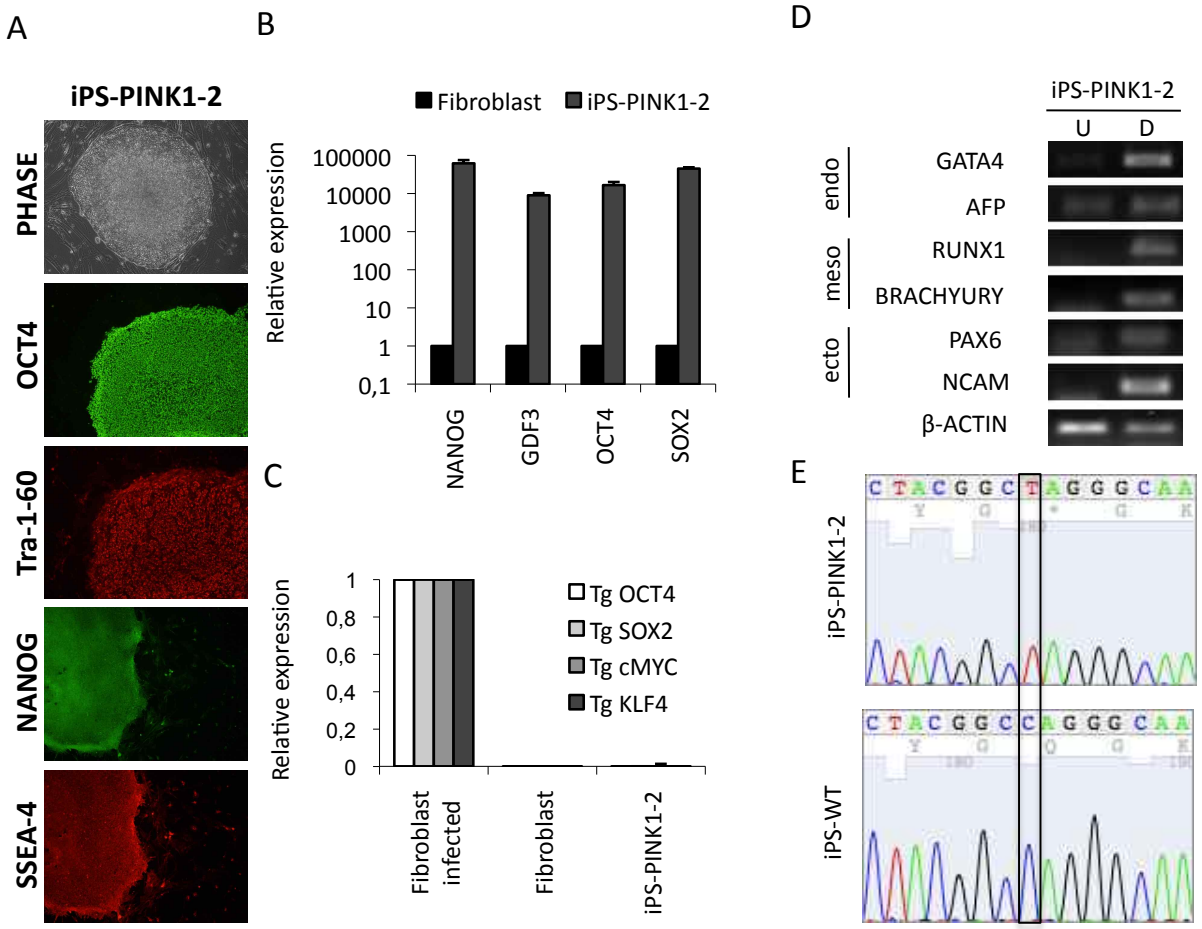

Fig. S3. Generation of iPS line (iPS-PINK1-2) from a family member of the PD patient described in Figure 1 (harboring the same PINK1 mutation c.1366C>T). **(A)** Immunofluorescence analysis shows the presence of pluripotency markers OCT4, Tra-1-60, NANOG and SSEA-4. **(B)** Expression levels of pluripotency markers NANOG, GDF3, OCT4 and SOX2 in fibroblasts and iPS cell lines relative to β-actin, as assessed by quantitative RT-PCR. The values from parental fibroblasts were set to 1. **(C)** Residual expression levels of transgenes OCT4, SOX2, cMYC and KLF4 (relative to β-actin) were examined by quantitative RT-PCR. The values from the infected fibroblasts (isolated 7 d post-infection) were set to 1. Uninfected fibroblasts were used as negative controls. The error bars indicate SD. **(D)** RT-PCR analyses of various differentiation markers for the three germ layers (endoderm: GATA4, AFP; mesoderm: RUNX1, BRACHYURY; ectoderm: PAX6, NCAM) in iPS cells that were undifferentiated (U) and after 4 days in suspension culture followed by 7 days in adherent culture. **(E)** Direct sequencing confirmed the PINK1 mutation c.1366C>T in iPS-PINK1-2.

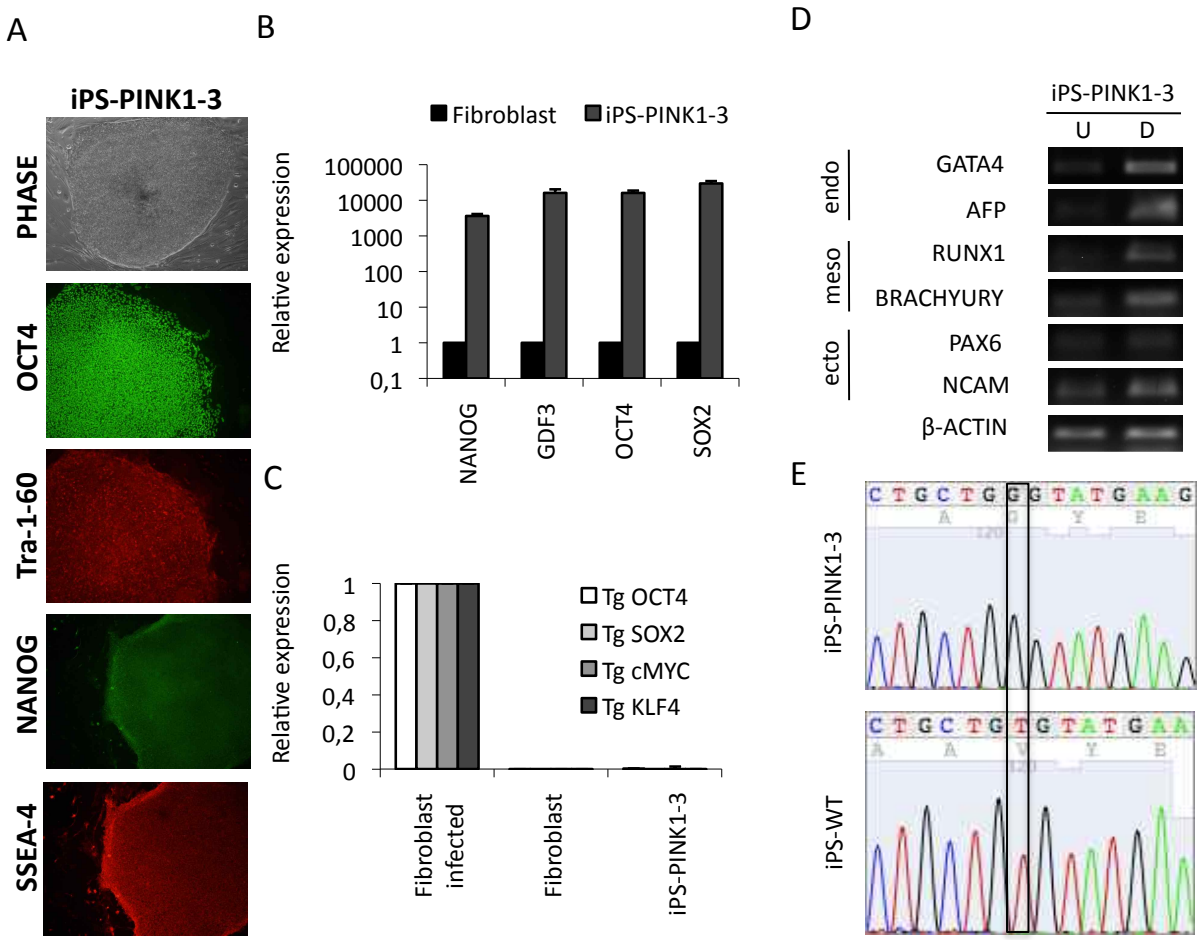

Fig. S4. Generation of iPS cell line (iPS-PINK1-3) from a PD patient harboring a missense mutation in the *PINK1* gene (c.509T>G; p.V170G). **(A)** Immunofluorescence analysis shows presence of pluripotency markers OCT4, Tra-1-60, NANOG and SSEA-4. **(B)** Expression levels of pluripotency markers NANOG, GDF3, OCT4 and SOX2 in fibroblasts and iPS cell lines relative to β-actin, as assessed by quantitative RT-PCR. The values from parental fibroblasts were set to 1. **(C)** Residual expression levels of transgenes OCT4, SOX2, cMYC and KLF4 (relative to β-actin) were examined by quantitative RT-PCR. The values from the infected fibroblasts (isolated 7 d post-infection) were set to 1. Uninfected fibroblasts were used as negative controls. The error bars indicate SD. **(D)** RT-PCR analyses of various differentiation markers for the three germ layers (endoderm: GATA4, AFP; mesoderm: RUNX1, BRACHYURY; ectoderm: PAX6, NCAM) in iPS cells that were undifferentiated (U) and after 4 days in suspension culture followed by 7 days in adherent culture. **(E)** Direct sequencing confirmed the *PINK1* mutation c.509T>G in iPS-PINK1-3.

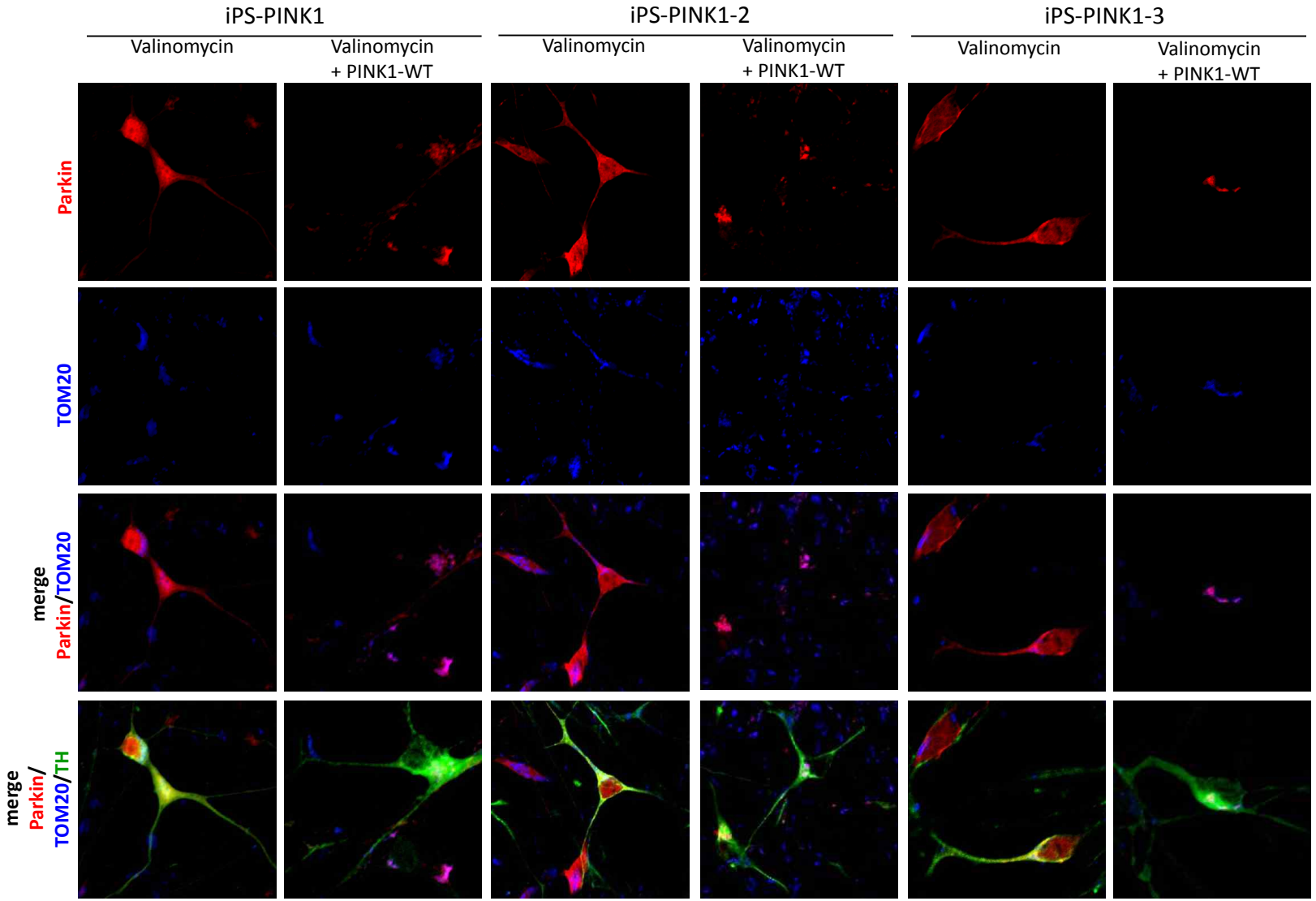

Fig. S5. Stress-induced mitochondrial translocation of Parkin is impaired in mutant PINK1 iPS cell-derived human DA neurons. Results are shown for the iPS mutant lines derived from three different patients. Neuronal cultures were infected with wild-type Parkin and treated with 1 µM valinomycin for 12h. After treatment, cells were fixed and immunostained with antibodies against Parkin (red), the mitochondrial marker TOM20 (blue) and the DA marker TH (green). Upon valinomycin treatment, Parkin fails to colocalize with mitochondria in DA neurons. Infection of PINK1 mutant neurons with wild-type PINK1 restored Parkin translocation to the mitochondria upon valinomycin treatment (see Figure 3 for details).

Tab. S1. Primer-sets used for PCR

Gene	Forward	Reverse
Klf4 endo[1]	GATGAACTGACCAGGCACTA	GTGGGTCATATCCACTGTCT
Oct4 endo[1]	CCTCACTTCACTGCACTGTA	CAGGTTTTCTTTCCCTAGCT
Sox2 endo[1]	CCCAGCAGACTTCACATGT	CCTCCCATTTCCCTCGTTTT
Klf4, Sox2, Oct4 trans[1]		CCTTGAGGTACCAGAGATCT
cMyc endo[1]	TGCCTCAAATTGGACTTTGG	GATTGAAATTCTGTGTAACTGC
cMyc trans[1]		CGCTCGAGGTTAACGAATT
GATA4 endoderm[1]	CTAGACCGTGGGTTTTGCAT	TGGGTTAAGTGCCCCTGTAG
AFP endoderm[1]	AGCTTGGTGGTGGATGAAAC	CCCTCTTCAGCAAAGCAGAC
BRACHYURY mesoderm[2]	AATTGGTCCAGCCTTGGAAT	CGTTGCTCACAGACCACA
RUNX1 mesoderm[1]	CCCTAGGGGATGTTCCAGAT	TGAAGCTTTTCCCTCTTCCA
NCAM ectoderm[1]	ATGGAAACTCTATTAAAGTGAACCTG	TAGACCTCATACTCAGCATTCCAGT
PAX6 ectoderm[2]	GTCCATCTTTGCTTGGGAAA	TAGCCAGGTTGCGAAGAACT
NANOG[1]	TGAACCTCAGCTACAAACAG	TGGTGGTAGGAAGAGTAAAG
GDF3[1]	AAATGTTTGTGTTGCGGTCA	TCTGGCACAGGTGTCTTCAG
PINK1 (c.1366 mutation)	GAGTTCAGATTAGCCCATGG	GACCTTCACTCTGGAACGAG
PINK1 (c.509 mutation)	GCTCACGGTGCATTCTTTTC	GCTTACCGAGATGTTCCACA
PCG-1α	TTGCCCAGATCTTCCTGAACTTG	CAAATGAGGGCAATCCGTCTTCA
PINK1	TTCCCCTTGGCCATCAAGA	ACCAGCTCCTGGCTCATTGT
mtDNA	AGGACAAGAGAAATAAGGCC	TAAGAAGAGGAATTGAACCTCTGACTGTAA

[1]Park et al., 2008c
[2]Huangfu et al., 2009