Supplementary Figure Legends

Supplementary Figure 1: Highly efficient transdifferentiation of MEFs into melanocyte-like cells by MITF (a) Brightfield images of ESCs prior to Dox induction and at day 12 post Dox induction. (b) MITF protein levels in MEFs at different time points post Dox induction. (c) *Mitf*, *Tyrp1*, *Tyrosinase*, *Trpm1*, and *Tyrp2* mRNA levels in MEFs at different time points post Dox induction compared to vehicle-treated cells. Levels were normalized to *Gapdh*, and fold changes relative to control are shown. Error bars represent ± SEM. * indicates $p < 0.05$ ($n = 3$). (d) MITF and TYR protein levels in *Mitf* knock in ESCs and *Mitf* knock in MEFs at day 12 post Dox induction (e) Immunostaining of MITF (green) and TYRP1 (green) in MEFs at day 12 post Dox induction. DAPI-stained nuclei appear blue. (f) Green pixel quantification for MITF-positive cells compared to DAPI-stained nuclei was made using ImageJ software and quantification for TYRP1-positive staining was made by counting the whole field. An average number of 30 cells were taken from each field, with two fields analyzed for each group.

Supplementary Figure 2: OCT4 transfection efficiency. MEFs were transfected with a plasmid for expression of OCT4 or empty vector as control. Left: Immunostaining of OCT4-flag (green) performed at day 6 post Dox induction. DAPI-stained nuclei appear blue (left panel). Right: *OCT4* mRNA level at day 6 post transfection. Levels were normalized to *Gapdh*, and fold changes relative to control are shown. Error bars represent ± SEM. * indicates $p < 0.05$ ($n = 3$). Error bars represent ± SEM.

Supplementary Figure 3: OCT4 interferes with MITF transcriptional activity. (a) Upper: HEK293T cells were co-transfected with luciferase reporter driven by the m*MLANA* promoter and with plasmids for expression of MITF, OCT4, or empty plasmid as control. Lower: WM3682 cells were co-transfected with luciferase reporter driven by the m*MLANA* promoter and with either OCT4 expression plasmid or empty plasmid as control. Luciferase activity was normalized to *Renilla* luciferase activity. Fold changes relative to control are shown. Error bars represent ± SEM, * indicates $p < 0.05$ ($n = 3$). (b) Upper: HEK293T cells were co-transfected with luciferase reporter driven by *TRPM1*, *TYR*, or *mMLANA* promoter and with plasmids for expression of

MITF, SOX2, or NANOG or empty plasmid as control. Lower: WM3682 cells were co-transfected with luciferase reporter driven by *TRPM1*, *TYR*, or m*MLANA* promoter and with SOX2 or NANOG expression plasmids or empty plasmid as control. Luciferase activity was normalized to *Renilla* luciferase activity. Fold changes relative to control are shown. Error bars represent ± SEM, * indicates $p < 0.05$ ($n = 3$). (c) Co-immunoprecipitation assay of MITF and OCT4 using protein extracts from melanoma WM3682 cells transfected with OCT4-flag. Samples were precipitated using anti-MITF antibody. Anti-flag was used for western blot. (d) A probe corresponding to the E-box region of the *TRPM1* promoter was used in EMSA to test *in vitro* binding of MITF to this sequence. Polyclonal anti-MITF antibody was used for supershift analysis. MITF bound probes and free probe are marked with arrows.

Supplementary Figure 4: *OCT4* and *MITF* are inversely correlated in melanoma cell lines and patients. (a) *MITF* and *OCT4* expression in melanoma cell lines. (b) Visualization of genetic alterations in melanoma related to MITF and OCT4 based on TCGA database using cBioPortal site for Cancer Genomics.

Supplementary Figure 5: OCT4, E2F7, or P53 and MITF overlapping ChIP-seq peaks. (a) Venn diagrams representing the number of unique gene promoters bound by E2F7 and MITF (ChIP-seq originated from melanoma cells) and the overlap between them based on ChIP-seq peaks. (b) A view of the selected biological pathways enriched within the overlapped gene set. (c) Venn diagrams representing the number of unique gene promoters and the overlap of MITF (ChIP-seq originated from normal melanocytes) with OCT4, P53, and E2F7. (d) Selected biological pathways identified by GO enrichment analysis of genes bound by both OCT4 and MITF.

Supplementary Figure 6: OCT4, SOX2, NANOG, and lineage transcription factor overlap. Right: Venn diagrams representing the number of gene promoters bound by OCT4, SOX2, NANOG, and the specified lineage transcription factors and the overlap between them based on ChIP-seq peaks . Left: Number of gene promoters that overlap between the combinations for each pair of OCT4, SOX2, and NANOG.

Supplementary Figure 7: Schematic illustration of the discussed cell fate conversions. Schematic representation of the roles of OCT4 and MITF in the three routes of cell fate conversions described herein**.**