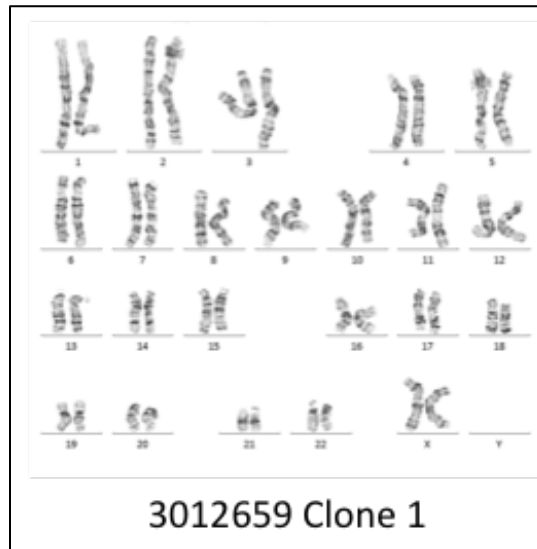


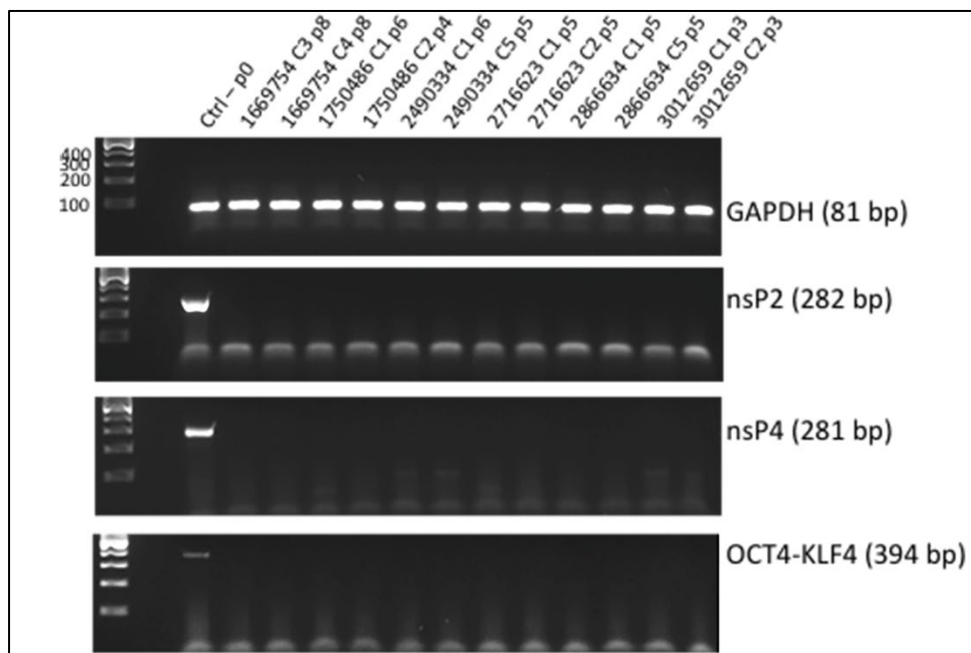
## Healthy control 1 – Cell Line ID: 278 (Patient ID: 3012659-MDPD1, age of sampling 68)

This cell line was obtained from Max Planck Institute for molecular Biomedicine, non-integrative reprogramming of human skin fibroblasts into iPSCs was done as previously described (Yoshioka et al. 2013) using VEE-OKSiG self-replicating RNA vectors, where Sox2 was replaced with an enhanced chimeric Sox factor (Velychko et al., manuscript in preparation).

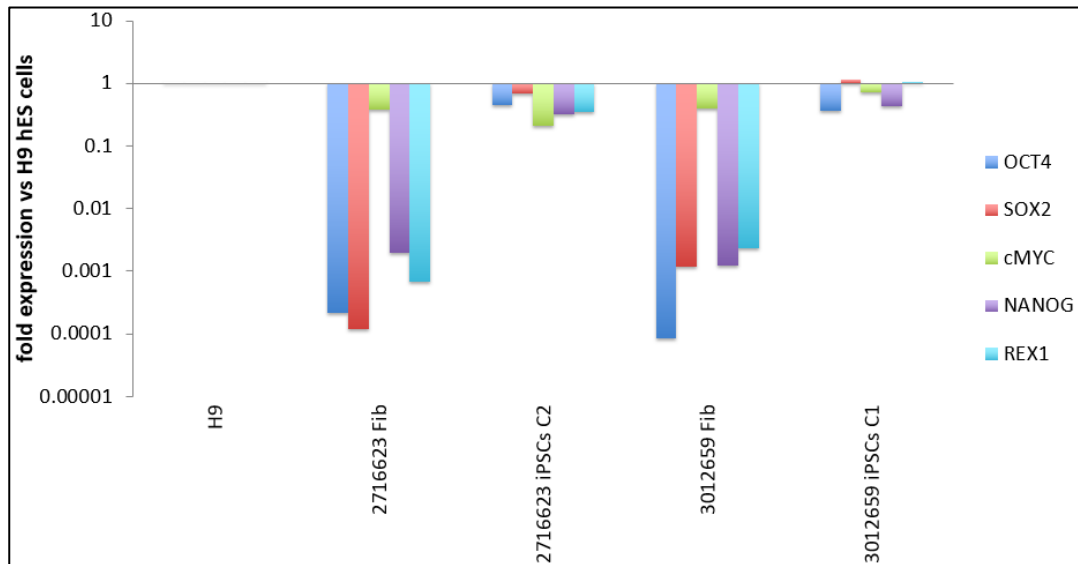
### 1) iPSC clone 3012659-C1 displays a normal diploid karyotype:



### 2) iPSC clones are free of transgenes: Total RNA was prepared from fibroblasts after transfection (Ctrl) and from iPSCs after three to eight passages and PCRs were conducted for the non-structural proteins nsP2 and nsP4 as well as for the OCT4-T2A-KLF4 region of the replicon and for GAPDH as loading control in order to check for persistent VEE RNA replicon.



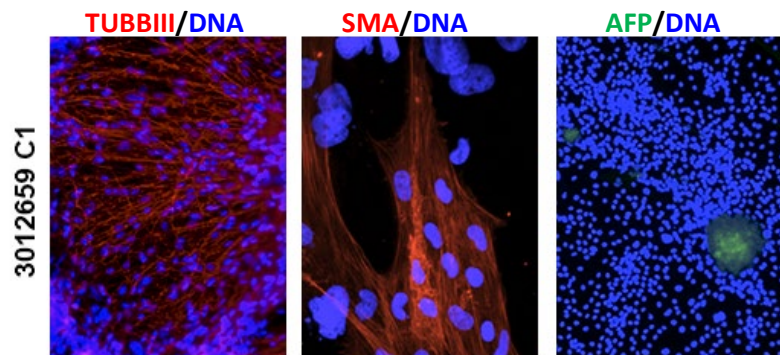
3) **Expression of pluripotency markers via qPCR:** The expression of indicated pluripotent marker genes as measured by qRT-PCR. The results are normalized to H9 hES cells.



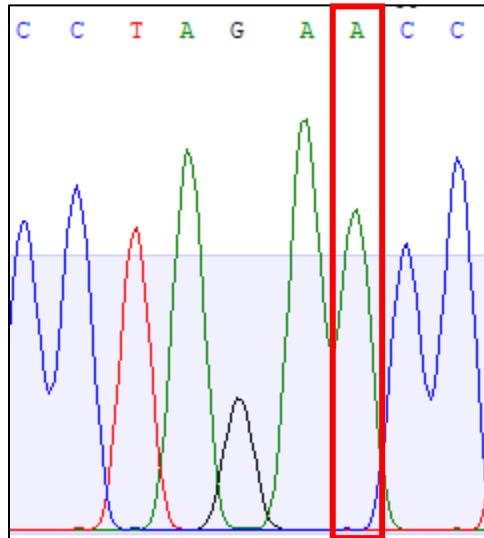
4) **Expression of pluripotency markers via Immunocytochemistry:** Immunofluorescence staining showed high expression of six pluripotency markers: Oct4 (green), SSEA-4 (red), Nanog (green) and TRA-1-60 (red). Nuclei were counterstained with DAPI (blue).



5) **Pluripotent differentiation potential was confirmed by immunocytochemical staining of EB-mediated differentiation of iPSC into cells of all three germ layers:** TUBBIII (red) was used as marker for ectoderm, SMA (red) as marker for mesoderm and AFP (green) as marker for endoderm.



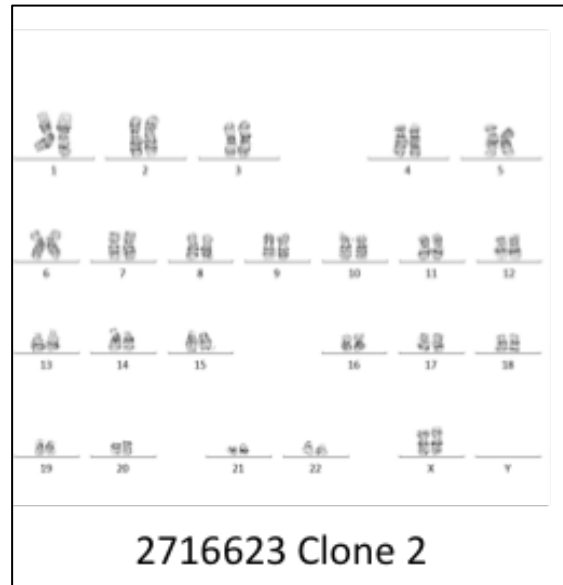
**6) Absence of the N370S mutation in the GBA gene:** Screening was done by extracting genomic DNA from blood samples using the GenElute™ Blood Genomic DNA Kit (Sigma, NA2020-1KT), PCR reactions were carried out using GoTaq® G2 Hot Start Master Mix (M7423, Promega). Primer sequences were F: TGTGTGCAAGGTCCAGGATCAG, R: ACCACCTAGAGGGGAAAGTG, which do not amplify the GBA pseudogene. Sample was sent for sequencing to Microsynth Seqlab.



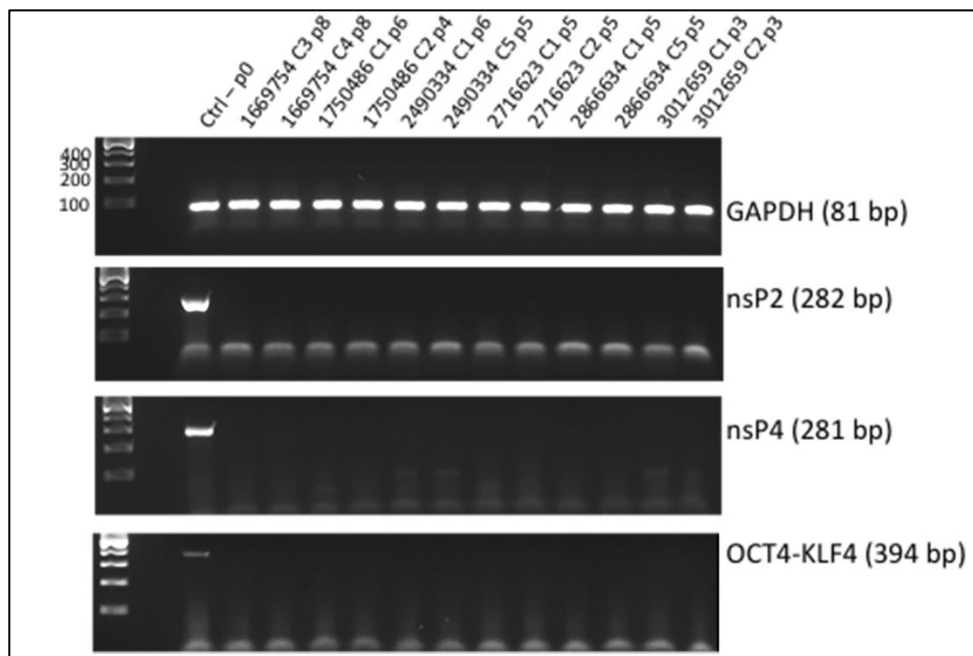
## Healthy control 2 – Cell Line ID: 277 (Patient ID: 2716623-MDPD1, age of sampling 65)

This cell line was obtained from Max Planck Institute for molecular Biomedicine, non-integrative reprogramming of human skin fibroblasts into iPSCs was done as previously described (Yoshioka et al. 2013) using VEE-OKSiG self-replicating RNA vectors, where Sox2 was replaced with an enhanced chimeric Sox factor (Velychko et al., manuscript in preparation).

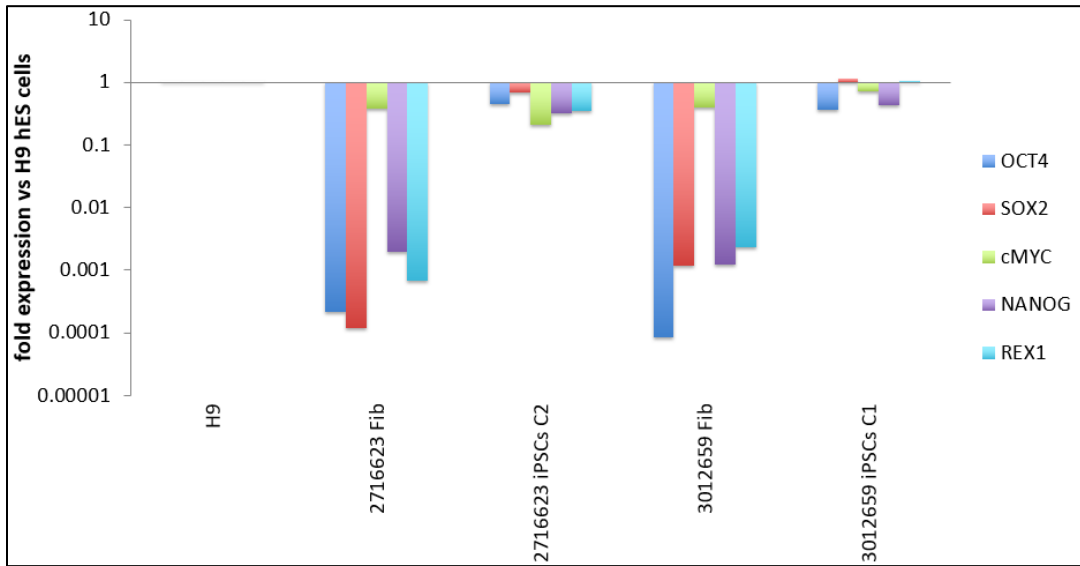
### 1) iPSC clone 2716623-C2 displays a normal diploid karyotype:



### 2) iPSC clones are free of transgenes: Total RNA was prepared from fibroblasts after transfection (Ctrl) and from iPSCs after three to eight passages and PCRs were conducted for the non-structural proteins nsP2 and nsP4 as well as for the OCT4-T2A-KLF4 region of the replicon and for GAPDH as loading control in order to check for persistent VEE RNA replicon.



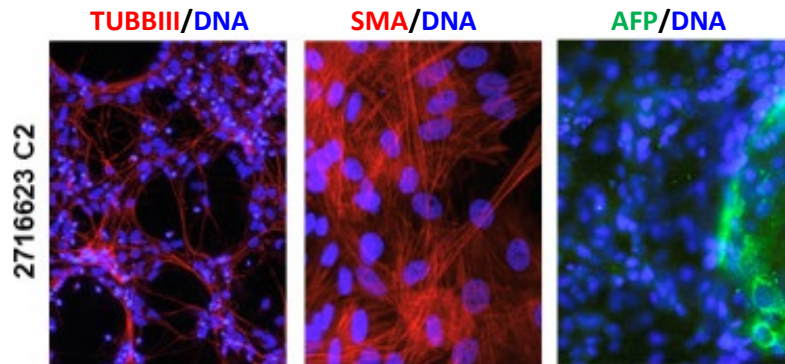
**3) Expression of pluripotency markers via qPCR:** The expression of indicated pluripotent marker genes as measured by qRT-PCR. The results are normalized to H9 hES cells.



**4) Expression of pluripotency markers via Immunocytochemistry:** Immunofluorescence staining showed high expression of six pluripotency markers: Oct4 (green), SSEA-4 (red), Nanog (green) and TRA-1-60 (red). Nuclei were counterstained with DAPI (blue).



**5) Pluripotent differentiation potential was confirmed by immunocytochemical staining of EB-mediated differentiation of iPSC into cells of all three germ layers:** TUBBIII (red) was used as marker for ectoderm, SMA (red) as marker for mesoderm and AFP (green) as marker for endoderm.





- 6) **Absence of the N370S mutation in the *GBA* gene:** Screening was done by extracting genomic DNA from blood samples using the GenElute™ Blood Genomic DNA Kit (Sigma, NA2020-1KT), PCR reactions were carried out using GoTaq® G2 Hot Start Master Mix (M7423, Promega). Primer sequences were F: TGTGTGCAAGGTCCAGGATCAG, R: ACCACCTAGAGGGGAAAGTG, which do not amplify the *GBA* pseudogene. Sample was sent for sequencing to Microsynth Seqlab.

