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Lab Resource: Stem Cell Line

Integration-free T cell-derived human induced pluripotent stem cells (iPSCs) from a patient with recessive dystrophic epidermolysis bullosa (RDEB) carrying two compound heterozygous mutations in the *COL7A1* gene

Munenari Itoh [a,*], Shiho Kawagoe [a], Katsuto Tamai [b], Hirotaka James Okano [c], Hidemi Nakagawa [a]

[a] Department of Dermatology, The Jikei University School of Medicine, Japan
[b] Department of Stem Cell Therapy Science, Graduate School of Medicine, Osaka University, Japan
[c] Division of Regenerative Medicine, The Jikei University School of Medicine, Japan

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ABSTRACT

Expanded human T cells from a Japanese female with recessive dystrophic epidermolysis bullosa (RDBE) were used to generate integration-free induced pluripotent stem cells (iPSCs) by exogenous expression of four reprogramming factors, *OCT3/4, SOX2, cMYC, KLF4*, using Sendai virus vector (SeVdp). The authenticity of established iPSC line, RDEB-iPSC26, was confirmed by the expressions of stem cell markers and the differentiation capability into three germ layer. RDEB-iPSC26 may be a useful cell resource for the establishment of in vitro RDEB modeling and the study for developing gene and cell therapy.

Resource table

Name of stem cell line	RDEB-iPSC26
Institution	Department of Dermatology, The Jikei University School of Medicine
Person who created resource	Munenari Itoh, Shiho Kawagoe
Contact person and email	Munenari Itoh: seafowl@jikei.ac.jp
Date archived/stock date	Dec 20, 2013
Origin	Human T cells
Type of resource	Biological reagent: human induced pluripotent stem cell (iPS); derived from a Japanese female with recessive dystrophic epidermolysis bullosa (RDEB)
Sub-type	Induced pluripotent stem cells (iPSCs)
Key transcription factors	*OCT3/4, SOX2, cMYC, KLF4*
Authentication	Identity and purity of cell line confirmed as shown in Fig. 1
Link to related literature	N/A
Information in public databases	Disease information; *OMIM #226600*
Ethics	Institutional ethics committee approval obtained (No. 23-271(6732)/Patient written informed consent obtained

1. Resource details

Recessive dystrophic epidermolysis bullosa (RDEB) is an inherited skin blistering disorder caused by mutations in the *COL7A1* gene encoding type VII collagen, the major component of anchoring fibrils at the basement membrane zone. RDEB is characterized by skin fragility, resulting in recurrent and incurable blisters. Since skin biopsy is borne by RDEB patients, we utilized peripheral blood cells for generating iPSCs. Expanded T cells isolated from a patient with RDEB were reprogrammed employing Sendai virus vectors (SeVdp) expressing four reprogramming factors, *OCT3/4, SOX2, cMYC, KLF4*. SeVdp is integration-free vector, and the absence of reprogramming genes in established iPSC line, RDEB-iPSC26, was confirmed by PCR analysis (Fig. 1A). The same pathogenic mutation in the *COL7A1* gene was also detected in genomic DNA isolated from RDEB-iPSC26 (Fig. 1B), proving the origin of cell source. The authenticity of RDEB-iPSC26 was confirmed by the followings; (1) the expression of stem cell markers by immunostaining (Fig. 1C) and RT-PCR (Fig. 1D), (2) the differentiation capability into three germ layers using in vitro differentiation through embryoid bodies (EBs) and teratoma formation (Fig. 1E). In addition, RDEB-iPSC26 maintains normal karyotype (46, XX) (Fig. 1F), and bisulfite sequencing revealed that the *NANOG* promoter region in RDEB-iPSC26 was unmethylated (Fig. 1G).

* Corresponding author.
E-mail address: seafowl@jikei.ac.jp (M. Itoh).

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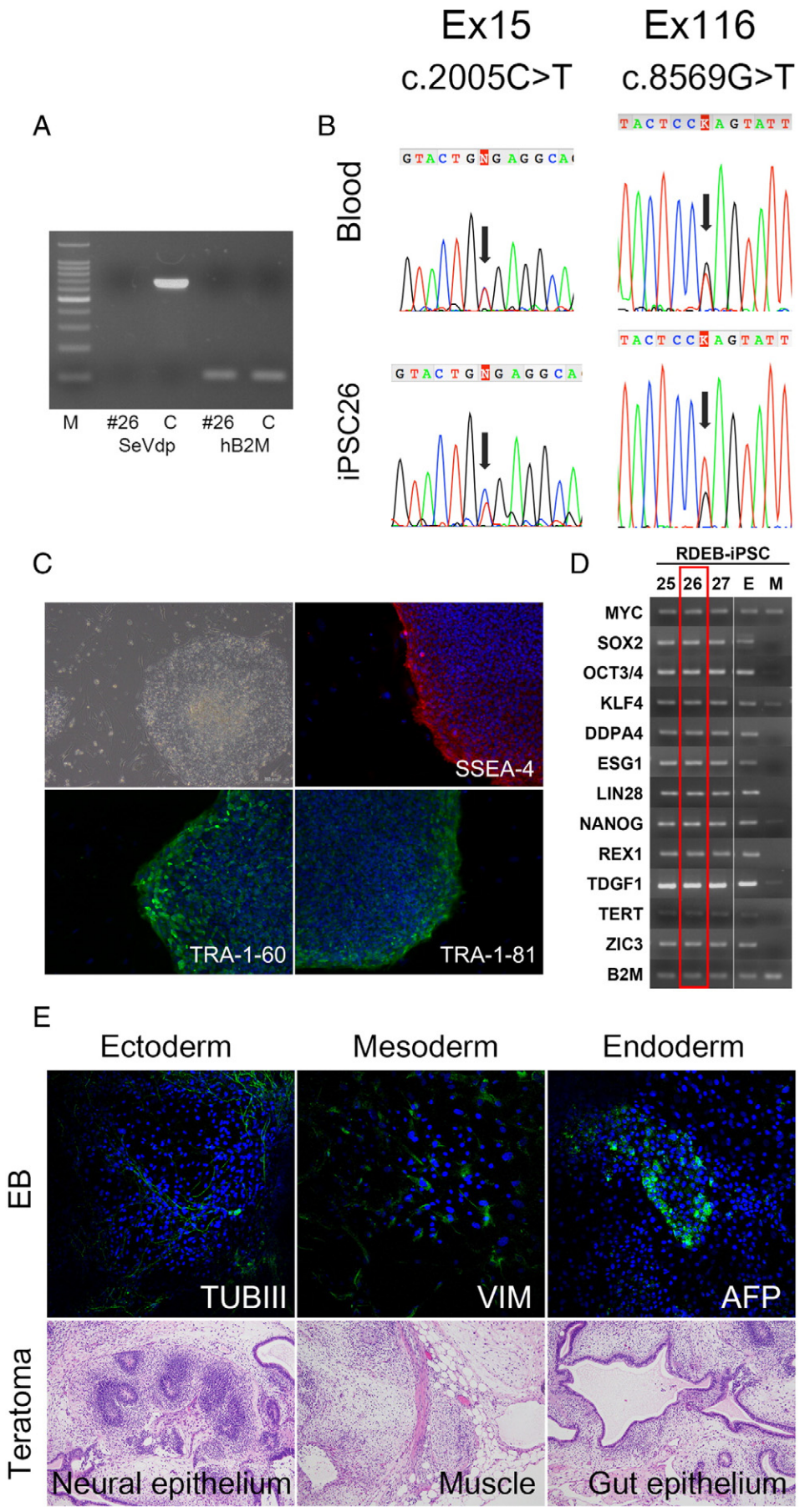
A
M
#26 SeVdp
C
#26 hB2M
C
B
Ex15
c.2005C>T
Ex116
c.8569G>T
Blood
iPSC26
C
SSEA-4
TRA-1-60
TRA-1-81
D
RDEB-iPSC
25 26 27 E M
MYC
SOX2
OCT3/4
KLF4
DDPA4
ESG1
LIN28
NANOG
REX1
TDGF1
TERT
ZIC3
B2M
E
Ectoderm
Mesoderm
Endoderm
EB
TUBIII
VIM
AFP
Teratoma
Neural epithelium
Muscle
Gut epithelium