Data Sheet

MOC22 Mouse Oral Squamous Cell Carcinoma (OSCC) Cell Line

Cancer Cell Line

SCC471

Pack Size: ≥ 1x10^6 viable cells/vial

Store in liquid nitrogen.

FOR RESEARCH USE ONLY

Not for use in diagnostic procedures. Not for human or animal consumption.

Background

Oral squamous cell carcinoma (OSCC) comprises 95% of all forms of head & neck cancer. The five-year survival rate for OSCC patients is approximately 50%; this poor patient outcome has not changed significantly in the last few decades. More recently, it has been suggested that immune cells may play a role in tumor-host interactions in tumorigenicity. However, the lack of available syngeneic mouse models has hindered progress. Xenograft models exist but require the use of immune-compromised mice and thus the necessary components of adaptive immunity are missing. The MOC (Mouse Oral Cancer) cell lines provide a syngeneic head and neck squamous cell carcinoma (HNSCC) model that can be transplanted in immune-competent C57BL/6 mice.[1] These cell lines are important models for the study of immune cell infiltration of tumors.

The MOC cell lines (MOC1, MOC2, MOC22) exhibit varying levels of *in vivo* tumor growth phenotypes. MOC1 and 22 have indolent growth phenotypes. MOC2 has a more aggressive *in vivo* growth phenotype – an injection of as few as 10,000 cells in mice is sufficient to form tumors. The more aggressive MOC2 line generates higher CD4$^+$ T cell levels. CD44, which has been implicated as a cancer stem cell marker that characterizes tumors with higher resistance to therapeutic treatment, is also expressed at a much higher level in MOC2 cells when compared to the MOC22 cell line. MOC22 cells present an indolent growth pattern which supports this data. Interestingly, the MOC22 cell line has also displayed higher average numbers of intratumoral CD8$^+$ T cells with and without anti-PD1 therapy. This cell line is especially useful in applications when comparing aggressive versus indolent phenotypes.

The MOC22 cell line includes HRAS and CASP8 driver mutations which are often associated together even though CASP8 mutations are uncommon in HNSCCs.[3] The MOC lines have been utilized to compare and identify potential clinical scenarios that are applicable to human Oral Squamous Cell Carcinoma. The MOC group of cell lines contained common HNSCC mutations in addition to human related OSCC driver pathways.

Source

Cell line was derived from a carcinogen-induced oral tumor in an immunocompetent C57BL/6 mouse.[1]

Short Tandem Repeat

M18-3: 15,16	M4-2: 20.3	M6-7: 15	M19-2: 13	M1-2: 19	M7-1: 26.2	M1-1: 15
M3-2: 14	M8-1: 16	M2-1: 16	M15-3: 22.3	M6-4: 19	M11-2: 16	M17-2: 15
M12-1: 17	M5-5: 17,18	MX-1: 27	M13-1: 17			

Quality Control Testing

- MOC22 cells are verified to be of mouse origin and negative for human, rat, Chinese hamster, Golden Syrian hamster, and nonhuman primate interspecies contamination, as assessed by a Contamination Clear panel by Charles River Animal Diagnostic Services.
- Cells tested negative for infectious diseases against a Mouse Essential CLEAR panel by Charles River Animal Diagnostic Services.
- Cells tested negative for mycoplasma.

Storage and Handling

MOC22 cells should be stored in liquid nitrogen until use. The cells can be cultured for at least 10 passages after initial thawing without significantly affecting the cell marker expression and functionality.

Representative Data

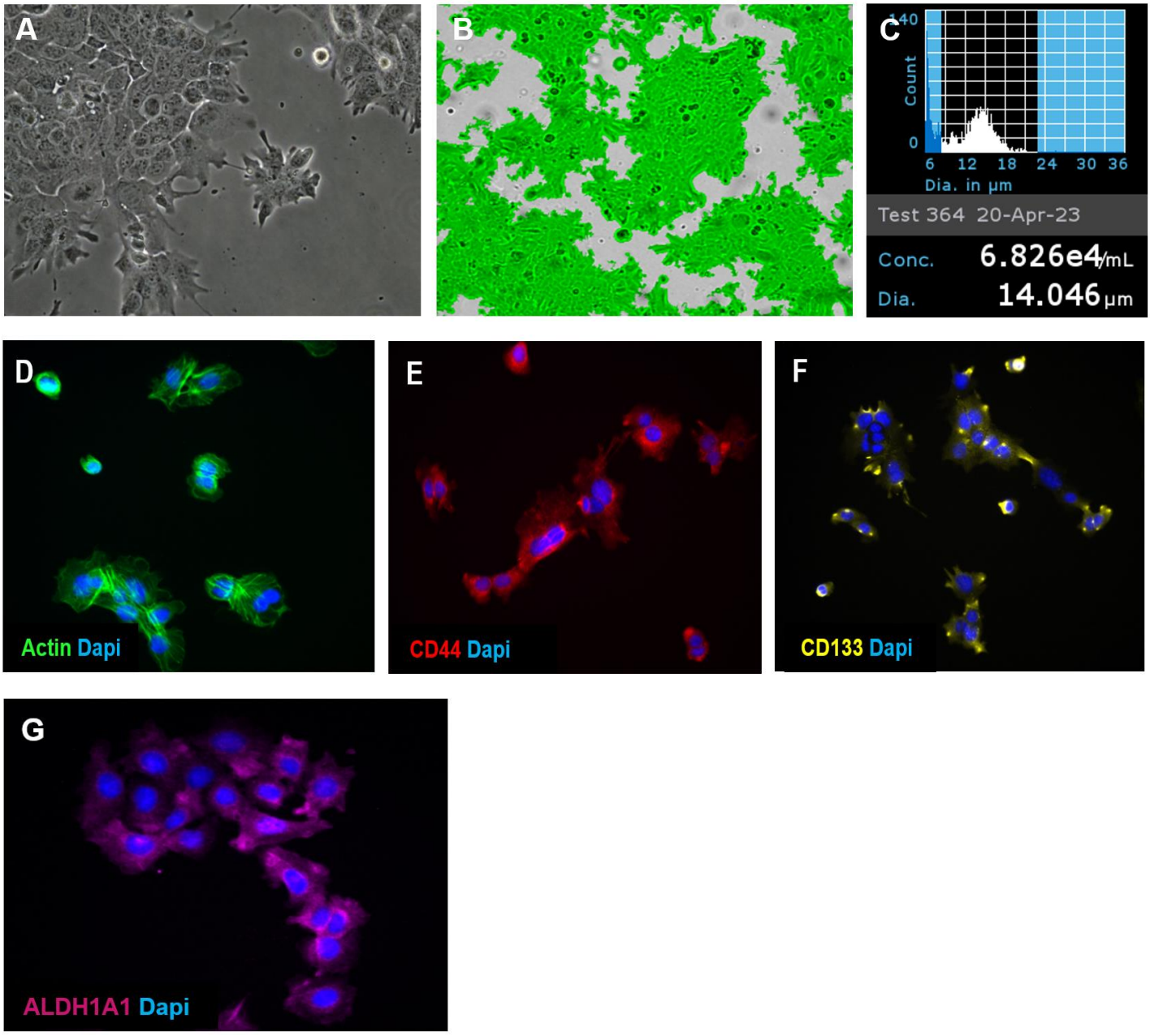

Figure 1. **A.** Brightfield image of MOC22 cells three days after thaw in a T75 flask. **B**. (MDCI10000) Cell confluency was assessed throughout the culture using the Millicell® Digital Cell Imager. **C.** (PHCC360KIT) Cell counting was performed using the Scepter™ 3.0 handheld automated cell counter using 60 µm sensors. **D.** (49409) Cells express actin, **E**. (SAB5700076) CD44, **F**. (Fisher Scientific PA5-38014) CD133 and **G.** (Fisher Scientific PA5-32127) ALDH1A1.

NOTE: Product catalog numbers indicated in ( ) can be purchased at SigmaAldrich.com unless otherwise stated.

Protocols

Thawing the Cells

MOC cell lines proliferate very rapidly. Upon thaw, it will take approximately 2-4 days for the cells to reach confluence. A passage ratio of 1:12 from an 80% confluent T175 flask) will take 2-4 days to reach 80% confluency.

1. Do not thaw the cells until the recommended medium is on hand. Cells can grow on standard tissue cultureware surfaces without any additional coating. Cells are thawed and expanded in MOC Expansion Medium comprising DMEM/F12 (DF-042-B) containing 10% FBS (ES-009-B), and 2 mM L-Glutamine (TMS-002-C).
2. Remove the vial of frozen MOC22 cells from liquid nitrogen and incubate in a 37 °C water bath. Closely monitor until the cells are completely thawed. Maximum cell viability is dependent on the rapid and complete thawing of frozen cells.

   IMPORTANT: Do not vortex the cells.
3. As soon as the cells are completely thawed, disinfect the outside of the vial with 70% ethanol. Proceed immediately to the next step.
4. In a laminar flow hood, use a 1- or 2-mL pipette to transfer the cells to a sterile 15 mL conical tube. Be careful not to introduce any bubbles during the transfer process.
5. Using a 10 mL pipette, slowly add dropwise 9 mL of MOC Expansion Medium (Step 1 above) to the 15 mL conical tube.

   IMPORTANT: Do not add the entire volume of media all at once to the cells. This may result in decreased cell viability due to osmotic shock.
6. Gently mix the cell suspension by slowly pipetting up and down twice. Be careful not to introduce any bubbles.

   IMPORTANT: Do not vortex the cells.
7. Centrifuge the tube at 300 x *g* for 2-3 minutes to pellet the cells.
8. Decant as much of the supernatant as possible. Steps 5-8 are necessary to remove residual cryopreservative (DMSO).
9. Resuspend the cells in 25–30 mL of MOC Expansion Medium.
10. Transfer the cell mixture to a T175 tissue culture flask.
11. Incubate the cells at 37 °C in a humidified incubator with 5% CO_2.

Subculturing the Cells

MOC22 cells are highly adherent and will require higher Accutase® volumes with longer incubation times.

1. Do not allow the cells to grow to confluency. MOC1 cells should be passaged at ~80-85% confluency.
2. Carefully remove the medium from the T175 tissue culture flask containing the 80% confluent layer of MOC1 cells.
3. Rinse the flask with 15 mL 1X PBS. Aspirate after the rinse.
4. Apply 10 mL of Accutase® and incubate in a 37 °C incubator for 10-15 minutes.
5. Inspect the flask and ensure the complete detachment of cells by gently tapping the side of the flask with the palm of your hand.

   NOTE: This cell line requires longer incubations and higher Accutase® volumes compared to most cancer cell lines.
6. Add 10 mL of MOC Expansion Medium to the plate.
7. Gently rotate the flask to mix the cell suspension. Transfer the dissociated cells to a 50 mL conical tube.
8. Centrifuge the tube at 300 x *g* for 3-5 minutes to pellet the cells.
9. Discard the supernatant, then loosen the cell pellet by tapping the tip of the tube with a finger.
10. Apply 2-5 mL of MOC Expansion Medium to the conical tube and resuspend the cells thoroughly. Large cell clumps may be broken up by gentle trituration.

    IMPORTANT: Do not vortex the cells.
11. Count the number of cells using a hemocytometer or a Scepter™ 3.0 handheld automated cell counter.
12. Plate the cells to the desired density. Typical split ratio is 1:10–1:12.

Cryopreservation of the Cells

MOC22 cells may be frozen in MOC Expansion Medium supplemented with 10% DMSO using a Nalgene® slow freeze Mr. Frosty™ container.

References

1. Judd NP, Allen CT, Winkler AE, Uppaluri R. 2012. Comparative analysis of tumor-infiltrating lymphocytes in a syngeneic mouse model of oral cancer. Otolaryngol Head Neck Surg. 147(3): 493–500.
2. Parikh A, Shin J, Faquin W, Lin DT, Tirosh I, Sunwoo JB, Puram SV. 2020. Malignant cell-specific CXCL14 promotes tumor lymphocyte infiltration in oral cavity squamous cell carcinoma. J Immunother Cancer. 8(2): e001048.
3. Onken MD, Winkler AE, Kanchi K-L, Chalivendra V, Law JH, Rickert CG, Kallogjeri D, Judd NP, Dunn GP, Piccirillo JF, et al. 2014. A surprising cross-species conservation in the genomic landscape of mouse and human oral cancer identifies a transcriptional signature predicting metastatic disease. Clin Cancer Res. 20(11): 2873–2884.

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