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Role of Threonines in the *Arabidopsis thaliana* Somatic Embryogenesis Receptor Kinase 1 Activation Loop in Phosphorylation*

Received for publication, March 16, 2001, and in revised form, August 6, 2001
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2001

Figure 4.
Green arrows: sharp vertical lines suggestive of splicing.
Red boxes: In Figure 4B, lanes 1 and 2 look unexpectedly similar to lanes 5 and 6, albeit shown at a different magnification.

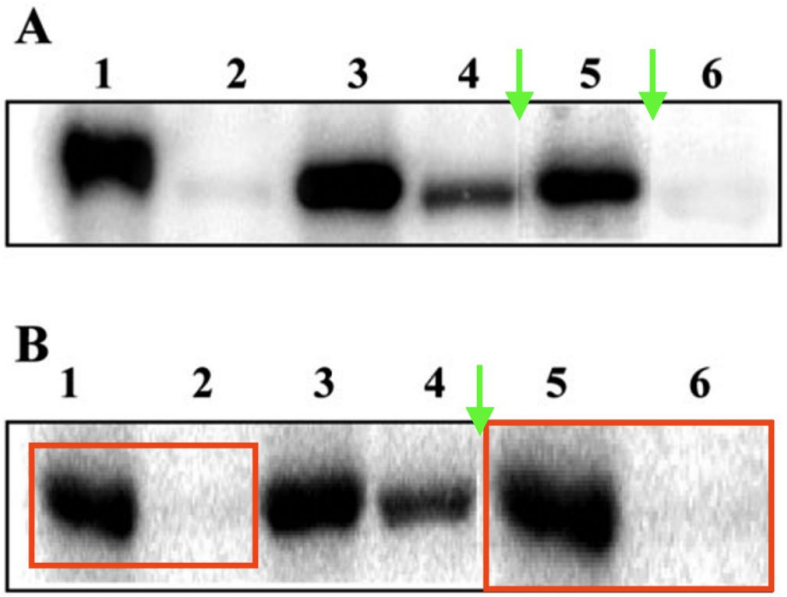


FIG. 4. **Autophosphorylation properties of AtSERK1^{kin} and AtSERK1^{kin} mutants.** Bacterially produced AtSERK1^{kin} proteins were purified, and aliquots of 500 ng were incubated with [γ-^{32}P]ATP as described under "Materials and Methods." After separation on 10% SDS-PAGE, the resulting gels were autoradiographed using a PhosphorImager. *A*, Autoradiographs of AtSERK1^{kin} (*lane 1*), AtSERK1$^{3T\rightarrow E}$ (*lane 2*), AtSERK1^{T459E} (*lane 3*), AtSERK1^{T462E} (*lane 4*), AtSERK1^{T463E} (*lane 5*) and AtSERK1^{T468E} (*lane 6*) proteins. *B*, Autoradiographs of AtSERK1^{kin} (*lane 1*), AtSERK1$^{3T\rightarrow E}$ (*lane 2*), AtSERK1^{T459A} (*lane 3*), AtSERK1^{T462A} (*lane 4*), AtSERK1^{T463A} (*lane 5*), and AtSERK1^{T468A} (*lane 6*) proteins.

doi:10.1006/jmbi.2001.4706 available online at http://www.idealibrary.com on IDEAL® J. Mol. Biol. (2001) 309, 641–655

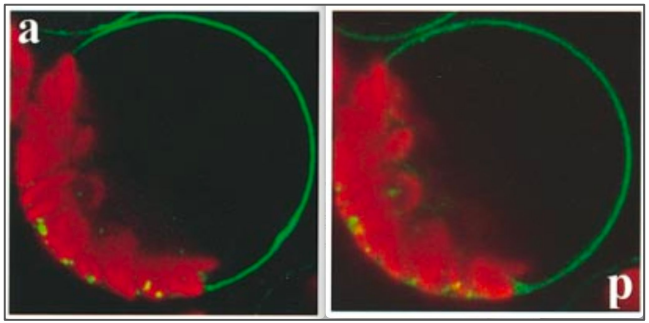
JMB

Subcellular Localization and Oligomerization of the *Arabidopsis thaliana* Somatic Embryogenesis Receptor Kinase 1 Protein

Khalid Shah, Theodorus W. J. Gadella Jr, Harrie van Erp, Valérie Hecht and Sacco C. de Vries*

2001

Concern about Figure 3:
Cyan boxes: Panels a (AtSERK1-YFP) and d (EGFRex-AtSERK1kin -YFP) appear to be showing the same specimens, albeit rotated 180 degrees and with slightly different green/red ratios. These photos are not completely identical but perhaps are two photos taken from the same specimen at different time points. Yet, the two panels are presented as different constructs.



Panels a and rotated-d compared -->

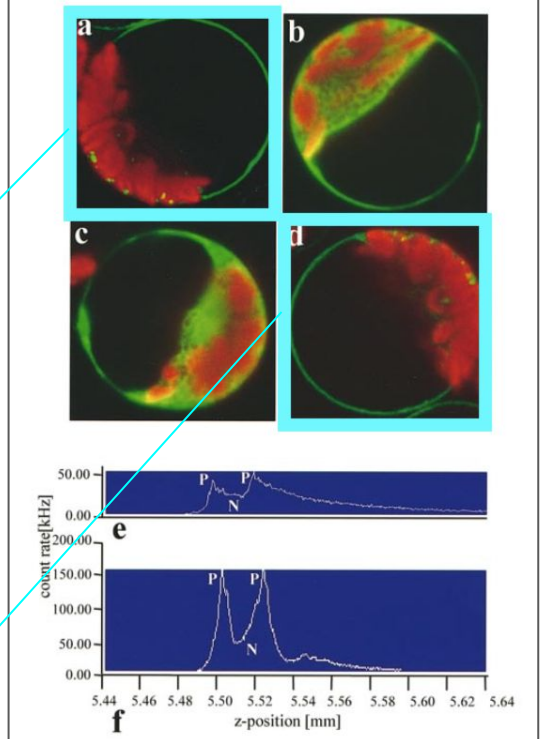


Figure 3. Confocal fluorescence images of protoplasts transfected with. (a) AtSERK1-YFP, (b) PMON999-YFP, (c) AtSERK1^{kin}-YFP, (d) EGFR^{ex}-AtSERK1^{kin}-YFP. In green the YFP fluorescence is shown and in red the chlorophyll fluorescence is shown. (e) and (f) Fluorescence correlation spectroscopy (FCS) of AtSERK1-YFP-CFP fusion proteins in insect cells. The profiles show the count rate along the optical *z*-axis of the (e) non-infected cells and the (f) AtSERK1-YFP expressing Sf21 cells. N and P indicate the fluorescence recorded in the nucleus and the plasma membrane, respectively.

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2001

Green boxes: Figure 4a (protoplasts transfected with LZ and the series of LRR truncated constructs AtSERK1deltaLZ-YFP) and Figure 7a (protoplast cotransfected with AtSERK1-YFP/CFP) appear to be showing the same photos. It appears that these photos represent different constructs. They are labeled differently but not sure if this duplication is appropriate or not.

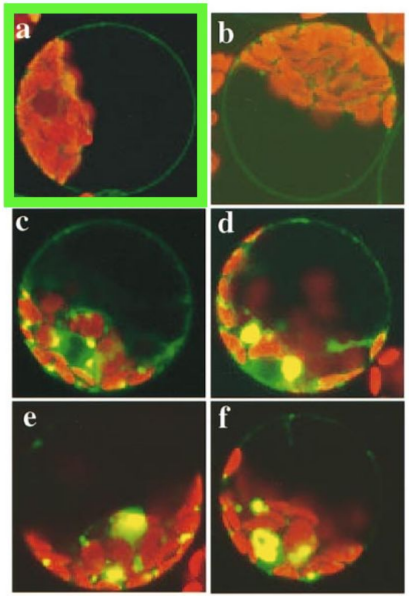


Figure 4. Confocal fluorescence images of protoplasts transfected with LZ and the series of LRR truncated constructs: (a) AtSERK1ΔLZ-YFP, (b) AtSERK1ΔLRR^{1}-YFP, (c) AtSERK1ΔLRR^{1,2}-YFP, (d) AtSERK1ΔLRR^{1,2,3}-YFP and (e) AtSERK1ΔLRR^{1,2,3,4}-YFP and (f) AtSERK1ΔLRR-YFP expression is shown. In green the YFP fluorescence is shown and in red the chlorophyll fluorescence is shown.

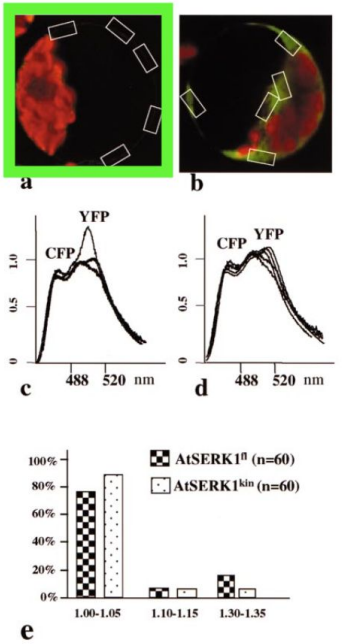


Figure 7. FSPIM analysis of fluorescent AtSERK1 fusion proteins. (a) Confocal image of a protoplast cotransfected with AtSERK1-YFP/CFP showing typical regions (rectangles) used for spectral measurements. (c) Emission spectra of the AtSERK1-CFP/YFP proteins obtained from the spectral images shown in (a). The *x*-axis represents wavelength (nm) and the *y*-axis represents the CFP and YFP fluorescence intensities. (b) and (d) The same for AtSERK1^{kin}-CFP/YFP, expressing protoplasts. (e) Comparison of the 525/475 nm fluorescence emission ratio (the YFP/CFP ratio), varying form 1.00 to 1.35, of the 60 protoplasts cotransfected with AtSERK1-CFP/YFP, and the protoplasts cotransfected with AtSERK1^{kin}-CFP/YFP.

Oncogene (2003) 22, 6865–6872

www.nature.com/onc

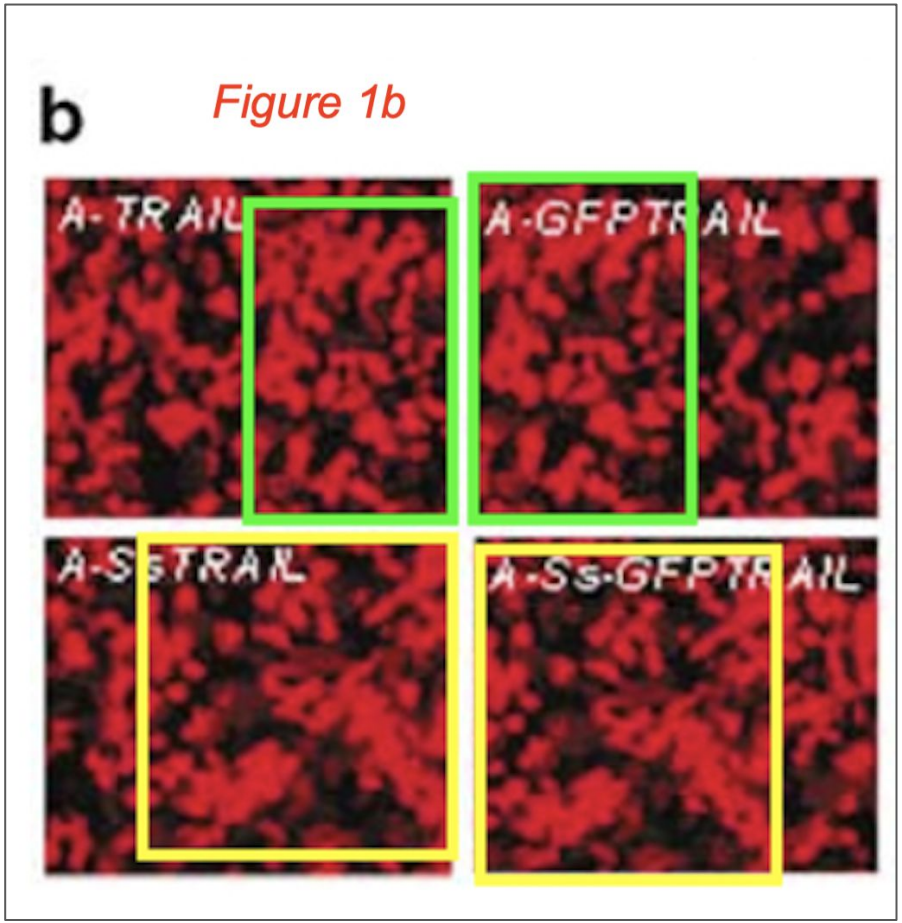
Real-time imaging of TRAIL-induced apoptosis of glioma tumors *in vivo*

Khalid Shah*[,1,2], Yi Tang[1], Xandra Breakefield[2] and Ralph Weissleder[1]

[1]*Center for Molecular Imaging Research, Massachusetts General Hospital, Harvard Medical School, Boston, MA, USA;* [2]*Molecular Neurogenetics Unit, Department of Neurology Massachusetts General Hospital, Harvard Medical School, Boston, MA, USA*

2003

Green boxes: The top two panels overlap, but represent different constructs.
Yellow boxes: The bottom two panels overlap, but represent different constructs.



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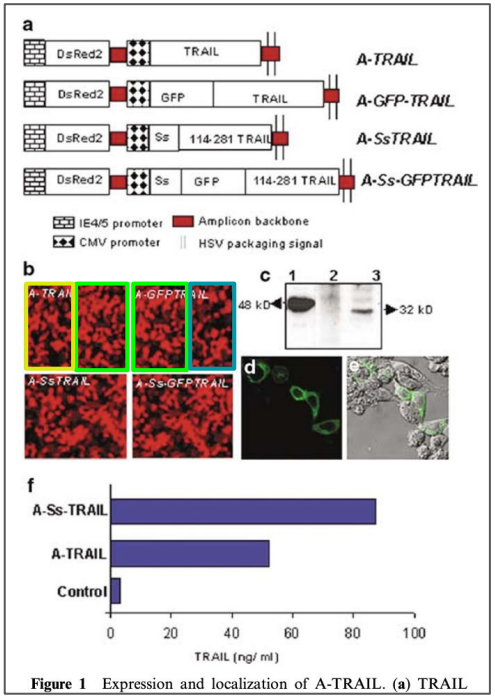

Figure 1 Expression and localization of A-TRAIL. (**a**) TRAIL

2004

[CANCER RESEARCH 64, 273–278, January 1, 2004]

In Vivo Imaging of HIV Protease Activity in Amplicon Vector-transduced Gliomas

Khalid Shah,[1,2] Ching-Hsuan Tung,[1] Chung-Hsun Chang,[1] Eric Slootweg,[2] Terence O'Loughlin,[1] Xandra O. Breakefield,[2] and Ralph Weissleder[1]

[1]*Center for Molecular Imaging Research, Department of Radiology and* [2]*Molecular Neurogenetics Unit, Department of Neurology, Massachusetts General Hospital, Harvard Medical School, Boston, Massachusetts*

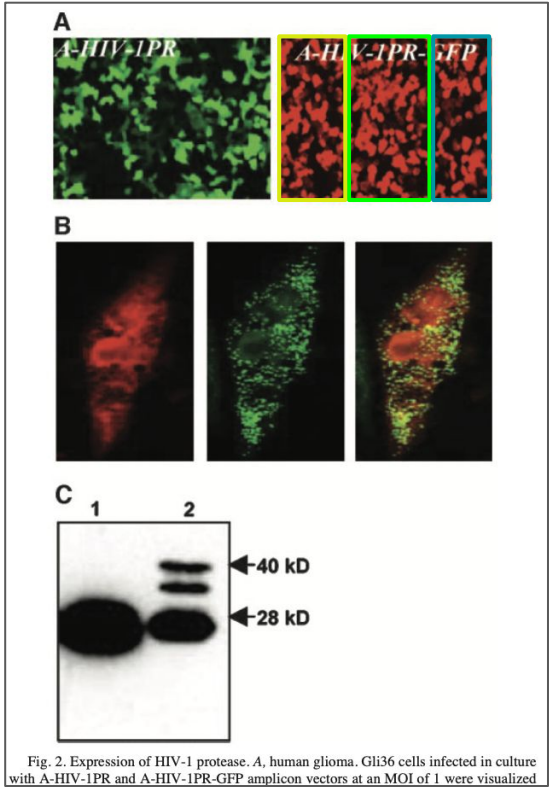

Fig. 2. Expression of HIV-1 protease. *A*, human glioma. Gli36 cells infected in culture with A-HIV-1PR and A-HIV-1PR-GFP amplicon vectors at an MOI of 1 were visualized

Green boxes:
The right panel in 2004's Figure 2A (right) overlaps with both top panels in Figure 1b in the 2003 paper (left), but appears to be representing a different experiment (firefly luciferase – luciferin in 2003 vs. HIV protease in 2004).

2003

Oncogene (2003) 22, 6865–6872

www.nature.com/onc

Real-time imaging of TRAIL-induced apoptosis of glioma tumors *in vivo*

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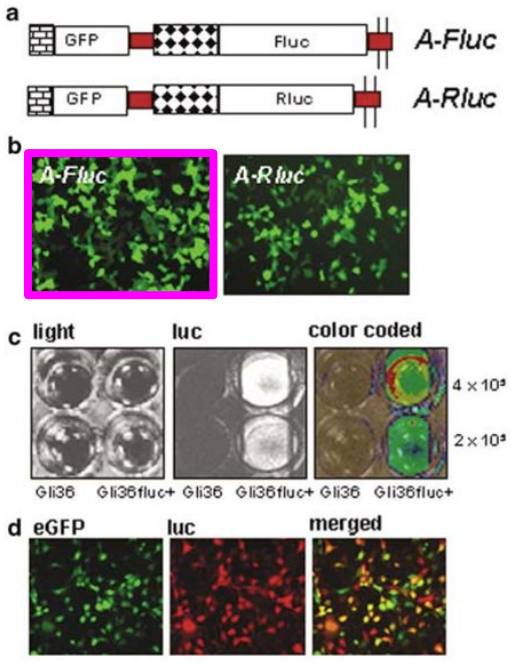

Figure 3 Characterization of Gli36fluc$^+$ cells. (**a**) Fluc and Rluc constructs in HSV-amplicon vectors (see Figure 1a) used to infect

2004

[CANCER RESEARCH 64, 273–278, January 1, 2004]

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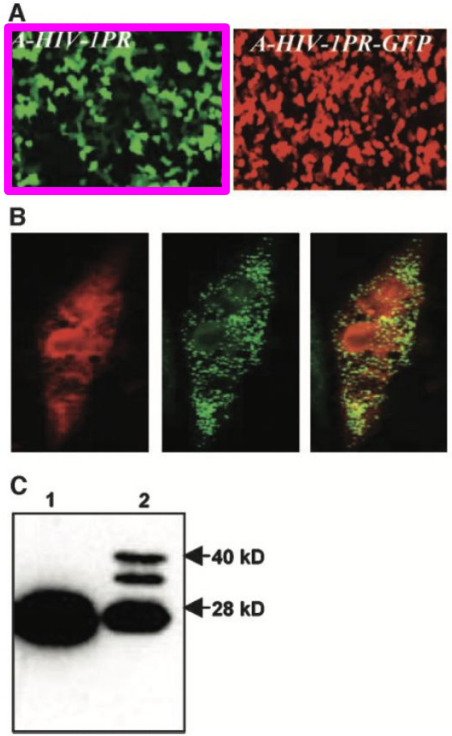


Fig. 2. Expression of HIV-1 protease. *A*, human glioma. Gli36 cells infected in culture with A-HIV-1PR and A-HIV-1PR-GFP amplicon vectors at an MOI of 1 were visualized

Pink boxes:
The green panel in 2004 Figure 2A (right) is identical to Figure 3b in the 2003 paper (left), but appears to be representing a different experiment (firefly luciferase – luciferin in 2003 vs. HIV protease in 2004).

2004

[CANCER RESEARCH 64, 3236–3242, May 1, 2004]

Inducible Release of TRAIL Fusion Proteins from a Proapoptotic Form for Tumor Therapy

Khalid Shah,[1,2] Ching-Hsuan Tung,[2] Katherine Yang,[1] Ralph Weissleder,[2] and Xandra O. Breakefield[1,2]

Molecular Neurogenetics Unit, [1]Department of Neurology and [2]Center for Molecular Imaging Research, Massachusetts General Hospital, Harvard Medical School, Boston, Massachusetts

Red boxes:
The non-infected panel in Figure 3A looks identical to the HSV-1PR infected panel in Figure 6B

Figure 3A

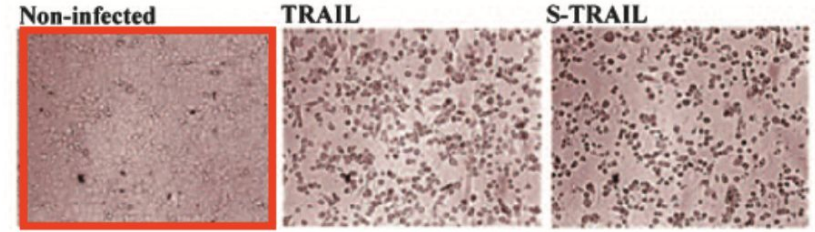
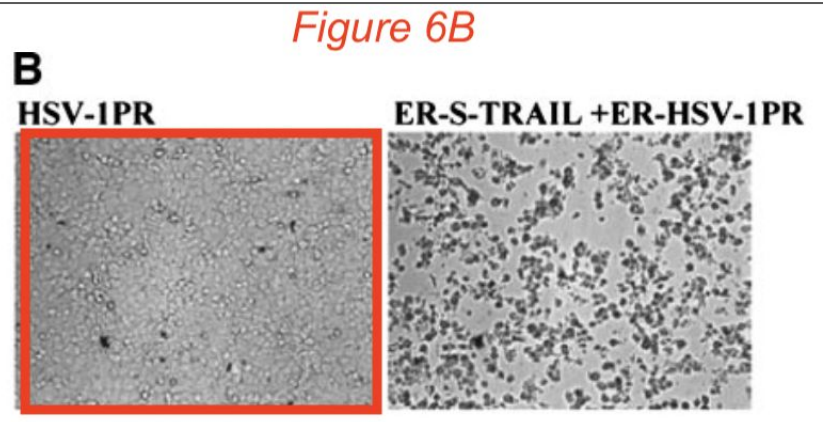


Figure 6B



2004

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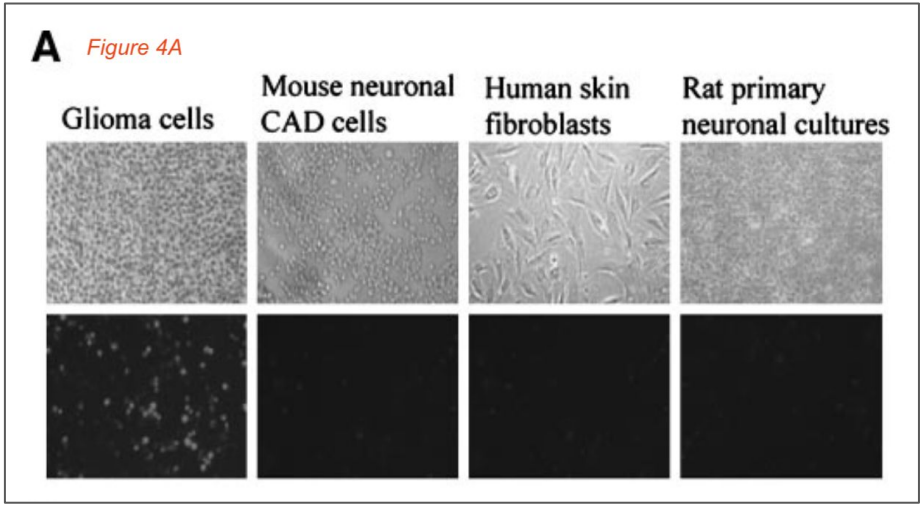
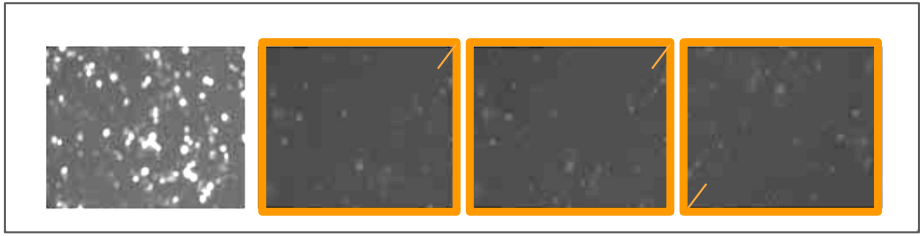
Orange boxes:
Three panels in the bottom row of Figure 4A look identical if the image brightness is adjusted to bring out the background. The bottom right image has been rotated 180 degrees.



Bottom row, made lighter



RESEARCH ARTICLE

Neoplasia • Vol. 9, No. 5, May 2007, pp. 435–442 435

www.neoplasia.com

Tumor Therapy Mediated by Lentiviral Expression of shBcl-2 and S-TRAIL[1]

Norman Kock[*,†,‡,2], *Randa Kasmieh*[*,†,2], *Ralph Weissleder*[†] *and Khalid Shah*[*,†]

*Department of Neurology, and †Center for Molecular Imaging Research (CMIR), Massachusetts General Hospital, Harvard Medical School, Boston, MA 02114, USA; ‡Department of Neurology, University of Lübeck, Lübeck, Germany

Figure 6.
Red and blue boxes: Both higher-magnification panels B and D match lower-magnification panel A, even though they represent different cells from differently transduced mice.

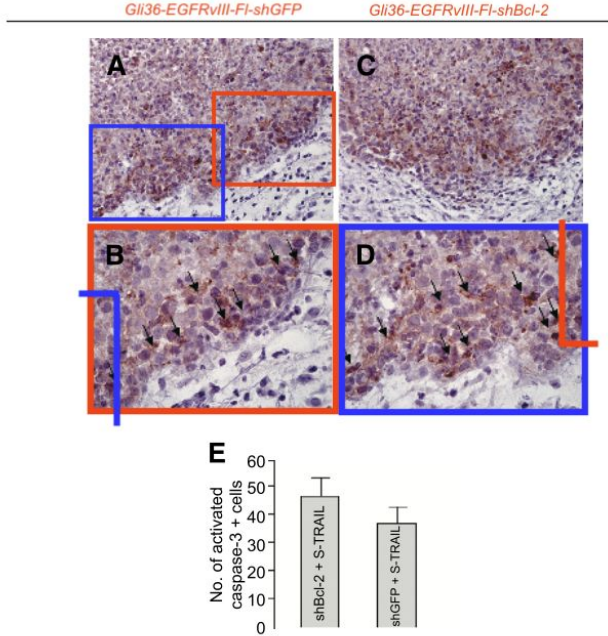


Figure 6. *Immunohistochemistry detects activated caspase-3 in glioma cells. Mice implanted with a mix of LV-S-TRAIL or control vector–transduced and nontransduced Gli36-EGFRvIII-FI-shGFP or Gli36-EGFRvIII-FI-shBcl-2 cells (Figure 5) were sacrificed on day 6 after tumor cell implantation, and tumors were sectioned and stained with anti–caspase-3 antibodies. The stained sections were counterstained with hematoxylin. (A and B) Sections from S-TRAIL–expressing Gli36-EGFRvIII-FI-shGFP gliomas. (C and D) S-TRAIL–expressing Gli36-EGFRvIII-FI-shBcl-2 gliomas. Caspase-3–stained cells are shown by arrows. (E) The number of activated caspase-3 positive cells was calculated by counting the positive cells in randomly selected field of views under a microscope. Original magnification, ×10 (A and C) and ×40 (B and D).*

2007

doi:10.1006/jmbi.2001.4706 available online at http://www.idealibrary.com on IDEAL® J. Mol. Biol. (2001) 309, 641–655

JMB

2001

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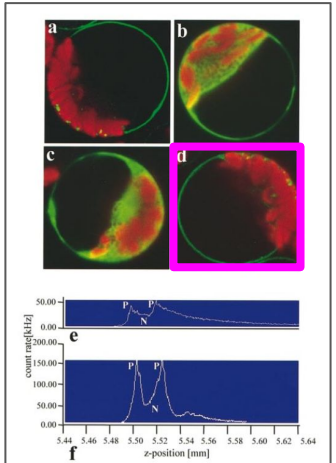


Figure 3. Confocal fluorescence images of protoplasts transfected with. (a) AtSERK1-YFP, (b) PMON999-YFP, (c) AtSERK1^{kin}-YFP, (d) EGFR^{ex}-AtSERK1^{kin}-YFP. In green the YFP fluorescence is shown and in red the chlorophyll fluorescence is shown. (e) and (f) Fluorescence correlation spectroscopy (FCS) of AtSERK1-YFP-CFP fusion proteins in insect cells. The profiles show the count rate along the optical z-axis of the (e) non-infected cells and the (f) AtSERK1-YFP expressing Sf21 cells. N and P indicate the fluorescence recorded in the nucleus and the plasma membrane, respectively.

Pink boxes:
Figure 1A of the 2008 paper shows the same panel as Figure 3d (or 3a) of the 2001 paper.
It is not clear if these represent the same construct or experiment.
Perhaps this is an appropriate duplication.

1052 2008 Biophysical Journal Volume 94 February 2008 1052–1062

Fluorescence Fluctuation Analysis of *Arabidopsis thaliana* Somatic Embryogenesis Receptor-Like Kinase and Brassinosteroid Insensitive 1 Receptor Oligomerization

Mark A. Hink,* Khalid Shah,* Eugenia Russinova,* Sacco C. de Vries,* and Antonie J. W. G. Visser*†
*MicroSpectroscopy Centre, Laboratory of Biochemistry, Wageningen University, 6703 HA Wageningen, The Netherlands;
and †Department of Structural Biology, Faculty of Earth and Life Sciences, Vrije Universiteit, 1081 HV Amsterdam, The Netherlands

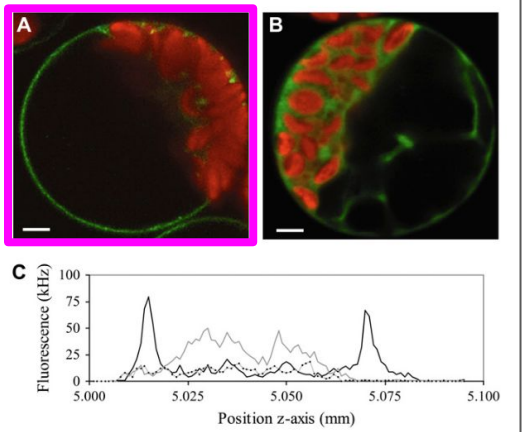


FIGURE 1 Expression of AtSERK1-ECFP. (*A*) Fluorescent confocal images of ECFP-labeled AtSERK1 protein expressed in cowpea protoplasts 16 h after transfection. CFP fluorescence, detected using a 480DF30 band-pass filter, is green in the false color-coded image, and chlorophyll fluorescence (LP650) is indicated by a red color. (*B*) AtSERK1^{kin}-ECFP. The confocal images were acquired in the equator of the protoplast by accumulating four subimages of 512 × 512 pixels with a focused laser beam of 458 nm set at 2.5 kW cm^{-2}. The bar represents 10 μm. (*C*) Lateral fluorescence intensity scans in the equator of cowpea protoplasts expressing AtSERK1-ECFP (*black line*) or AtSERK1^{kin}-ECFP (*gray line*). The intensity profile of nontransfected cells is indicated by the dotted line.

2008

4406 • The Journal of Neuroscience, April 23, 2008 • 28(17):4406–4413

Cellular/Molecular

Bimodal Viral Vectors and *In Vivo* Imaging Reveal the Fate of Human Neural Stem Cells in Experimental Glioma Model

Khalid Shah,[1,2,5] **Shawn Hingtgen,**[1] **Randa Kasmieh,**[1] **Jose Luiz Figueiredo,**[1] **Elisa Garcia-Garcia,**[4] **Alberto Martinez-Serrano,**[4] **Xandra Breakefield,**[2] **and Ralph Weissleder**[1,3,5]

[1]Center for Molecular Imaging Research, Department of Radiology, [2]Department of Neurology, and [3] Center for Systems Biology, Department of Systems Biology, Massachusetts General Hospital, Harvard Medical School, Boston, Massachusetts 02115, [4]Departamento de Biología Molecular, Centro de Biología Molecular Severo Ochoa, Consejo Superior de Investigaciones Científicas, Universidad Autónoma de Madrid, 28049 Madrid, Spain, and [5]Harvard Stem Cell Institute, Harvard University, Cambridge, Massachusetts 02138

Cyan boxes:
In Figure 1B, the Stem cells / Fluc-DsRed2 and Rluc-DsRed2 panels appear to overlap. These are presented as different constructs in Figure 1A.

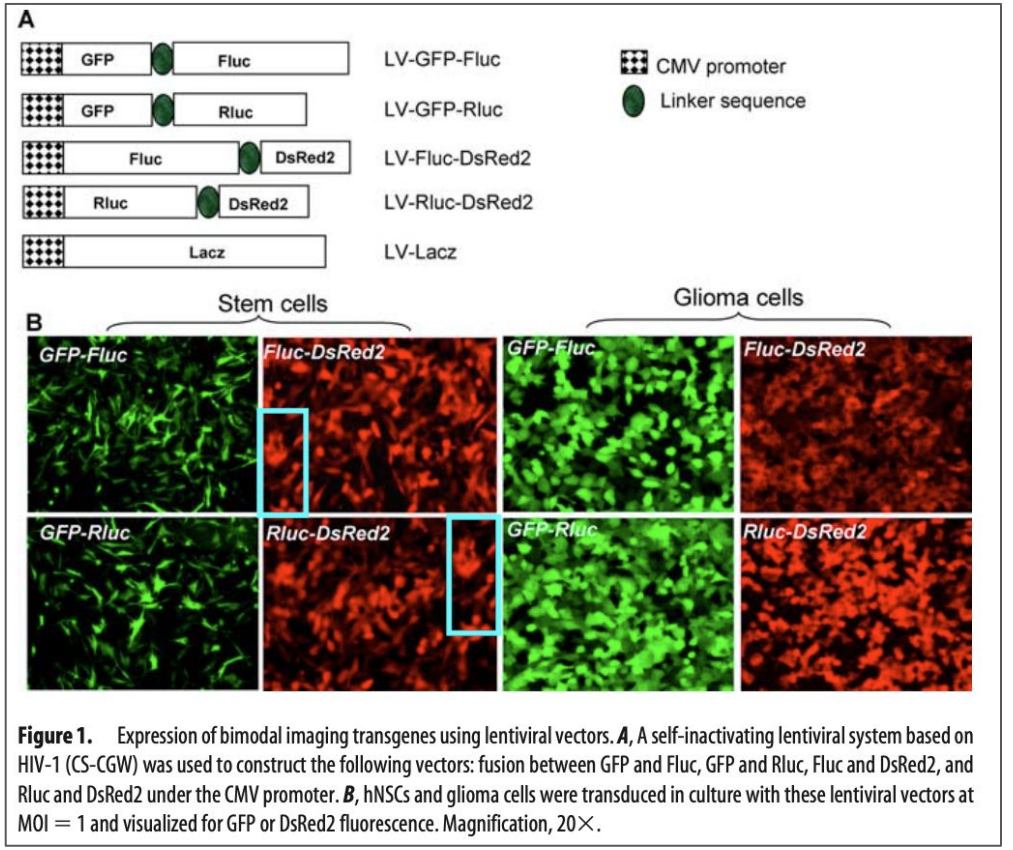


Figure 1. Expression of bimodal imaging transgenes using lentiviral vectors. ***A***, A self-inactivating lentiviral system based on HIV-1 (CS-CGW) was used to construct the following vectors: fusion between GFP and Fluc, GFP and Rluc, Fluc and DsRed2, and Rluc and DsRed2 under the CMV promoter. ***B***, hNSCs and glioma cells were transduced in culture with these lentiviral vectors at MOI = 1 and visualized for GFP or DsRed2 fluorescence. Magnification, 20×.

PNAS

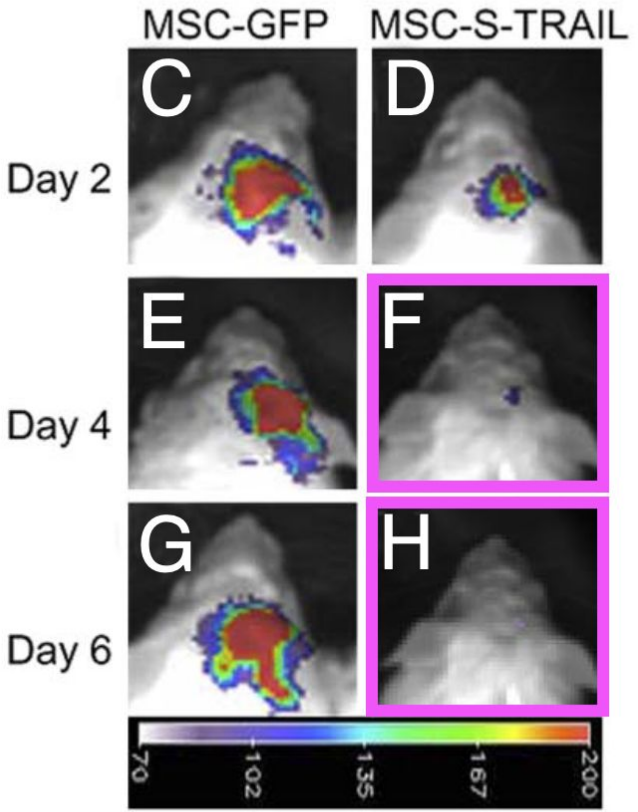
Assessment of therapeutic efficacy and fate of engineered human mesenchymal stem cells for cancer therapy

Laura S. Sasportas[a,b,1], Randa Kasmieh[a,b,1], Hiroaki Wakimoto[c], Shawn Hingtgen[a,b], Jeroen A. J. M. van de Water[a,b], Gayatry Mohapatra[e], Jose Luiz Figueiredo[b], Robert L. Martuza[c], Ralph Weissleder[b,f], and Khalid Shah[a,b,d,2]

[a]Molecular Neurotherapy and Imaging Laboratory, [b]Center for Molecular Imaging Research (CMIR), Department of Radiology, Departments of [c]Neurosurgery, [d]Neurology, and [e]Pathology, and [f]Center for Systems Biology, Department of Systems Biology, Massachusetts General Hospital, Harvard Medical School Boston, MA 02114

Figure 4:
Pink boxes: Panels F (Day 4) and H (Day 6) look remarkably similar. It is unlikely that the mouse was put in the exact same position two days later. The bioluminescence signals are different.

Figure 4, detail



2009

2010

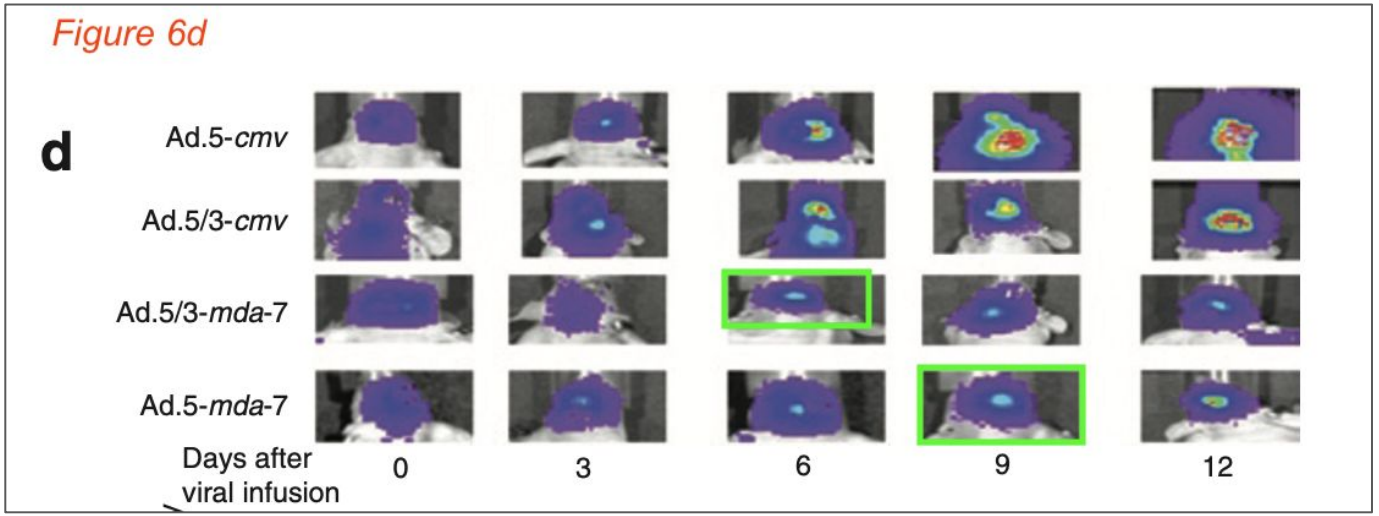
original article

Molecular Therapy vol. 18 no. 6 june 2010

Inhibition of Multiple Protective Signaling Pathways and Ad.5/3 Delivery Enhances *mda*-7/IL-24 Therapy of Malignant Glioma

Hossein A Hamed[1], Adly Yacoub[1], Margaret A Park[1], Patrick J Eulitt[1], Rupesh Dash[2], Devanand Sarkar[2,3], Igor P Dmitriev[4], Maciej S Lesniak[5], Khalid Shah[6], Steven Grant[1,3,7,8], David T Curiel[4], Paul B Fisher[2,3,8] and Paul Dent[1,3,8]

Figure 6d.
Green boxes: Two panels representing different animal groups and time points look more similar than expected, albeit stretched and cropped differently.



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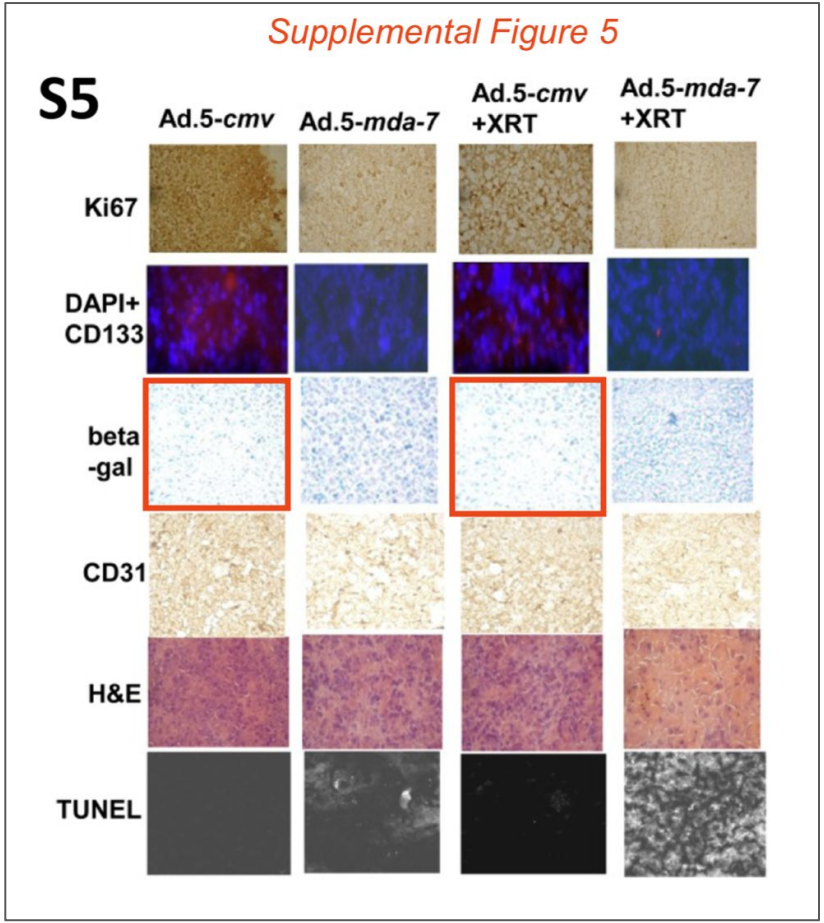
Supplemental Figure 5.
Red boxes: Two beta-gal panels representing different animal groups look identical.



2010

original article

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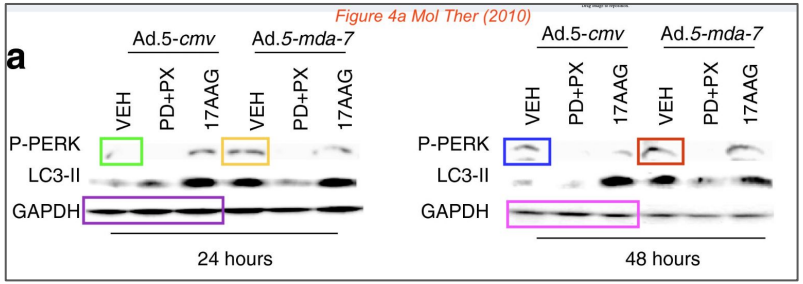
Molecular Therapy vol. 18 no. 6 june 2010

Inhibition of Multiple Protective Signaling Pathways and Ad.5/3 Delivery Enhances *mda*-7/IL-24 Therapy of Malignant Glioma

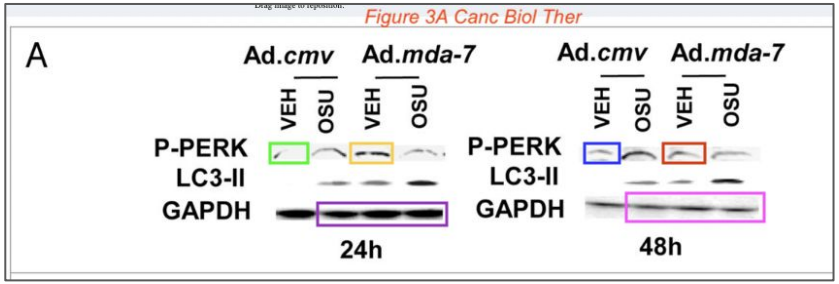
Hossein A Hamed[1], Adly Yacoub[1], Margaret A Park[1], Patrick J Eulitt[1], Rupesh Dash[2], Devanand Sarkar[2,3], Igor P Dmitriev[4], Maciej S Lesniak[5], Khalid Shah[6], Steven Grant[1,3,7,8], David T Curiel[4], Paul B Fisher[2,3,8] and Paul Dent[1,3,8]



Cancer Biology & Therapy 9:7, 526-536; April 1, 2010; © 2010 Landes Bioscience

OSU-03012 enhances Ad.*mda*-7-induced GBM cell killing via ER stress and autophagy and by decreasing expression of mitochondrial protective proteins

Hossein A. Hamed,[1] Adly Yacoub,[1] Margaret A. Park,[1] Patrick Eulitt,[1] Devanand Sarkar,[3,4] Igor P. Dimitriev,[5] Ching-Shih Chen,[6] Steven Grant,[1,2,4] David T. Curiel,[5] Paul B. Fisher[3,4] and Paul Dent[1,4,*]



Boxes of the same color highlight bands that appear to have been used in both papers, suggesting that lanes were copy/pasted. In both papers, the p-PERK lanes that look duplicated represent the Vehicle, so these might be the same experiments, but the GAPDH lanes do not represent the same experiments.
Part of these issues have been raised on PubPeer by 'Peer 1' on July 2016.

nature neuroscience

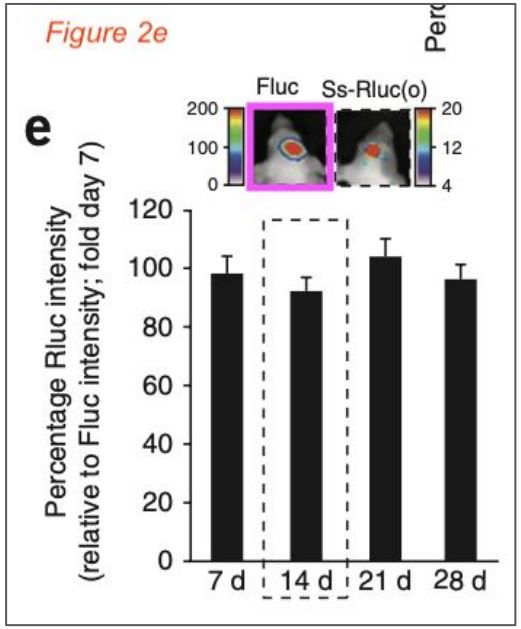
ARTICLES

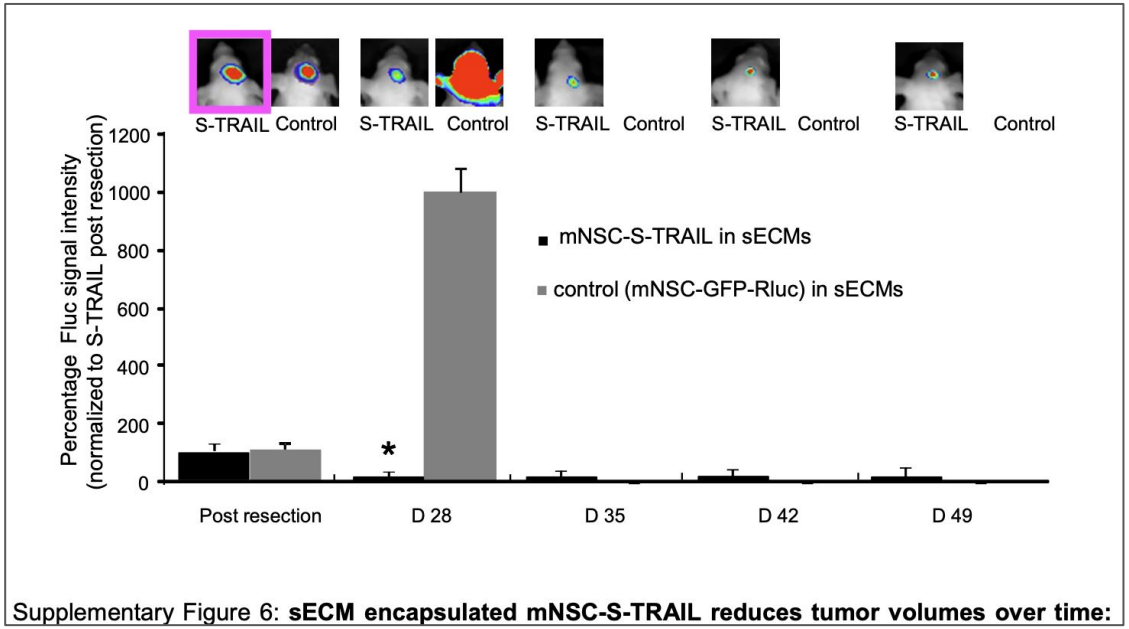
Encapsulated therapeutic stem cells implanted in the tumor resection cavity induce cell death in gliomas

Timo M Kauer[1,2], Jose-Luiz Figueiredo[1,2], Shawn Hingtgen[1,2] & Khalid Shah[1–4]

2012

Pink boxes: Panels in Figure 2e (Fluc) and Suppl Figure 6 (S-TRAIL) look unexpectedly similar, while the labels suggest these are different experiments.





Supplementary Figure 6: **sECM encapsulated mNSC-S-TRAIL reduces tumor volumes over time:**

2012

OPEN ACCESS Freely available online

PLOS ONE

Evaluating the Effect of Therapeutic Stem Cells on TRAIL Resistant and Sensitive Medulloblastomas

Irina Nesterenko[1,2☯], Simone Wanningen[1,2☯], Tugba Bagci-Onder[1,2], Maarten Anderegg[1,2], Khalid Shah[1,2,3,4*]

1 Molecular Neurotherapy and Imaging Laboratory, Massachusetts General Hospital, Harvard Medical School, Boston, Massachusetts, United States of America, **2** Department of Radiology, Massachusetts General Hospital, Harvard Medical School, Boston, Massachusetts, United States of America, **3** Department of Neurology, Massachusetts General Hospital, Harvard Medical School, Boston, Massachusetts, United States of America, **4** Harvard Stem Cell Institute, Harvard University, Cambridge, Massachusetts, United States of America

Cyan boxes:
The UW429+hMSC-GFP panel in Figure 2d looks remarkably similar to the hMSC-S-TRAIL panel in Figure 2e, albeit stretched differently

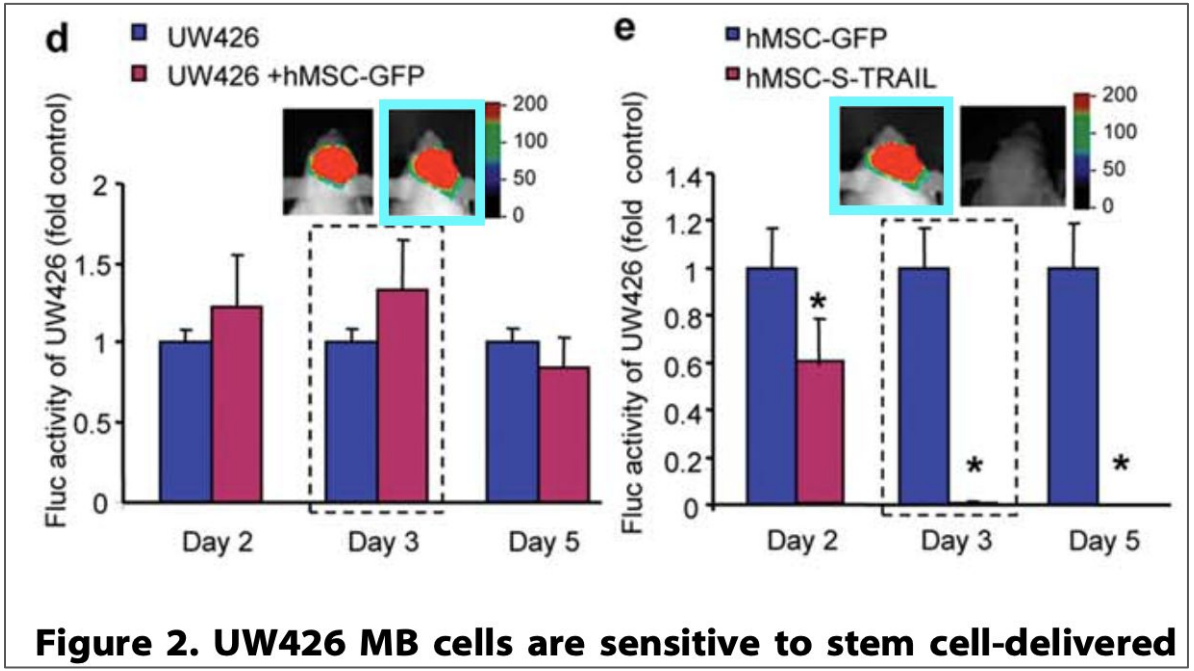


Figure 2. UW426 MB cells are sensitive to stem cell-delivered

2012

OPEN ACCESS Freely available online

PLOS ONE

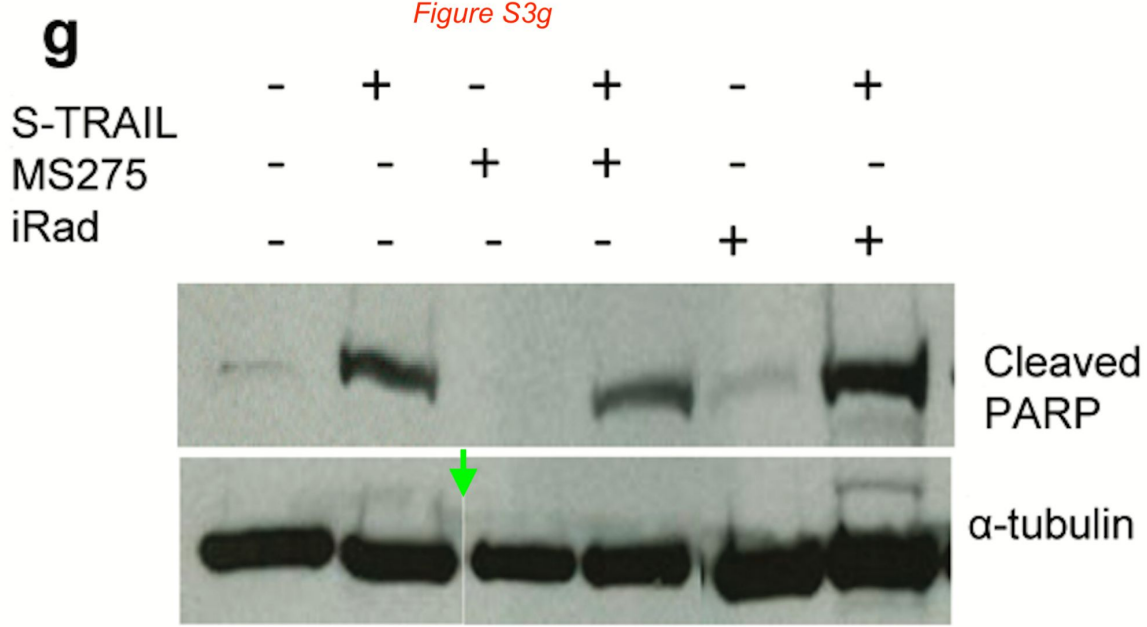
Evaluating the Effect of Therapeutic Stem Cells on TRAIL Resistant and Sensitive Medulloblastomas

Irina Nesterenko[1,2☯], Simone Wanningen[1,2☯], Tugba Bagci-Onder[1,2], Maarten Anderegg[1,2], Khalid Shah[1,2,3,4*]

1 Molecular Neurotherapy and Imaging Laboratory, Massachusetts General Hospital, Harvard Medical School, Boston, Massachusetts, United States of America, **2** Department of Radiology, Massachusetts General Hospital, Harvard Medical School, Boston, Massachusetts, United States of America, **3** Department of Neurology, Massachusetts General Hospital, Harvard Medical School, Boston, Massachusetts, United States of America, **4** Harvard Stem Cell Institute, Harvard University, Cambridge, Massachusetts, United States of America

Green arrow:
The tubulin blot appears to have a differential splice, i.e., a splice not visible in the corresponding position in the cleaved PARP blot.

Figure S3g



2012

PNAS

Therapeutic stem cells expressing variants of EGFR-specific nanobodies have antitumor effects

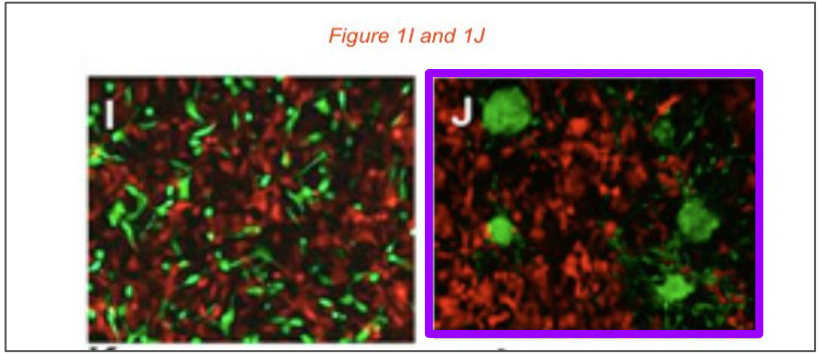
Jeroen A. J. M. van de Water[a,b,c], Tugba Bagci-Onder[a,b], Aayush S. Agarwal[a,b], Hiroaki Wakimoto[a,b,d], Rob C. Roovers[c], Yanni Zhu[a,b], Randa Kasmieh[a,b], Deepak Bhere[a,b], Paul M. P. Van Bergen en Henegouwen[c], and Khalid Shah[a,b,e,f,1]

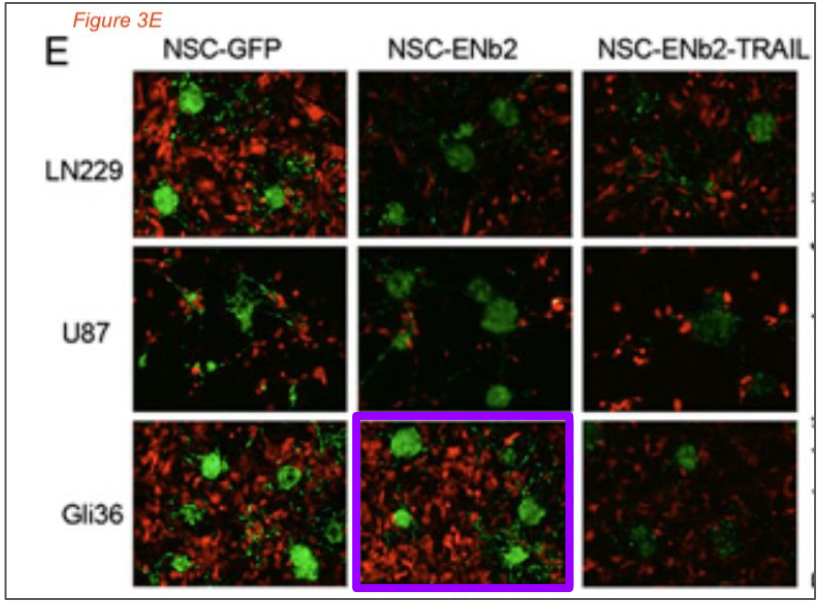
[a]Molecular Neurotherapy and Imaging Laboratory, and Departments of [b]Radiology, [e]Neurology, and [d]Neurosurgery, Massachusetts General Hospital, Harvard Medical School, Boston, MA 02129; [c]Cell Biology, Department of Biology, Science Faculty, Utrecht University, 3508, Utrecht, The Netherlands; and [f]Harvard Stem Cell Institute, Harvard University, Cambridge, MA 02138

Edited by Webster K. Cavenee, Ludwig Institute for Cancer Research, University of California at San Diego, La Jolla, CA, and approved August 28, 2012 (received for review February 17, 2012)

Purple boxes: Two panels in Figures 1J and 3E, respectively, look the same but appear to be representing different co-cultures.

Figure 1J: 'Photomicrographs showing cocultured hNSC (I) and mNSC (J) expressing ENb2 (green) and LN229 GBM cells (red)'
Figure 3E: ' FLuc-mCherry expressing GBM cells (LN229, U87, and Gli36) cocultured with mouse NSC expressing GFP (control), ENb2, or ENb2-TRAIL'





2012

Therapeutic stem cells expressing variants of EGFR-specific nanobodies have antitumor effects

Jeroen A. J. M. van de Water[a,b,c], Tugba Bagci-Onder[a,b], Aayush S. Agarwal[a,b], Hiroaki Wakimoto[a,b,d], Rob C. Roovers[c], Yanni Zhu[a,b], Randa Kasmieh[a,b], Deepak Bhere[a,b], Paul M. P. Van Bergen en Henegouwen[c], and Khalid Shah[a,b,e,f,1]

[a]Molecular Neurotherapy and Imaging Laboratory, and Departments of [b]Radiology, [e]Neurology, and [d]Neurosurgery, Massachusetts General Hospital, Harvard Medical School, Boston, MA 02129; [c]Cell Biology, Department of Biology, Science Faculty, Utrecht University, 3508, Utrecht, The Netherlands; and [f]Harvard Stem Cell Institute, Harvard University, Cambridge, MA 02138

Edited by Webster K. Cavenee, Ludwig Institute for Cancer Research, University of California at San Diego, La Jolla, CA, and approved August 28, 2012 (received for review February 17, 2012)

Blue boxes: Panels C and I in Figure 4 overlap, albeit with different aspect ratios, but appear to be representing different co-cultures.

'(C–E) Photomicrographs show presence of NSC (green) within U87-mCherry-Fluc tumors (Red).'

'(F–K) Photomicrographs of H&E stained and fluorescence microscopy analyzed sections of the brain of GBM-bearing mice treated with NSC-GFP (F and I)'

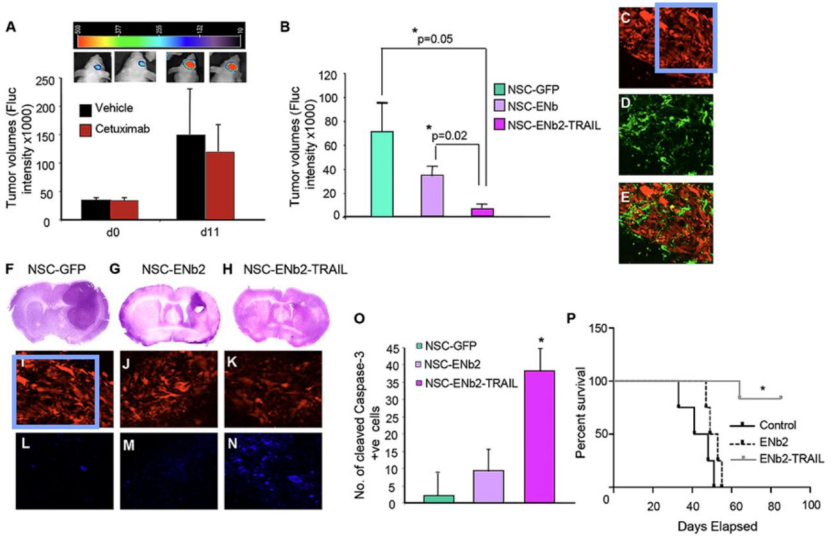


Fig. 4. In vivo efficacy of ENb2 and ENb2-TRAIL secreting NSC on GBM volumes. (*A*) Tumor volumes measured by FLuc bioluminescence imaging signal intensity of nude mice bearing U87-mCherry-FLuc intracranial tumors and injected with Cetuximab (1 mg per mouse·d^{-1}) or vehicle daily for 1 wk. (*B*) Tumor volumes of nude mice bearing established intracranial U87-mCherry-FLuc tumors treated with NSC expressing GFP, ENb2, or ENb2-TRAIL. (*C–E*) Photomicrographs show presence of NSC (green) within U87-mCherry-Fluc tumors (Red). (*F–K*) Photomicrographs of H&E stained and fluorescence microscopy analyzed sections of the brain of GBM-bearing mice treated with NSC-GFP (*F* and *I*), NSC-ENb2 (*G* and *J*), and NSC-ENb2-TRAIL (*H* and *K*) showing the changes in tumor volumes and mCherry+ tumor cells. (*L–O*) Photomicrographs (*L–N*) and plot (*O*) showing the extent of cleaved caspase-3 staining (blue) in brain sections of NSC-GFP (*L*), NSC-ENb2 (*M*), and NSC-ENb2-TRAIL (*N*) treated mice. Plot shows the number of cleaved caspase-3 cells in different treatment groups (*O*). (Original magnification: 20x.) (*P*) Kaplan–Meier survival curves of mice bearing established tumors and implanted with NSC expressing GFP, ENb2, or ENb2-TRAIL intratumorally ($n = 5$ per group). For *A* and *B*, data were represented as mean ± SEM, and * denotes $P < 0.05$, Student's *t* test. For *P*, * denotes $P < 0.05$ as compared Enb2 and control groups, log-rank test.

STEM CELLS
STEM CELLS 2013;31:1706–1714
TRANSLATIONAL AND CLINICAL RESEARCH

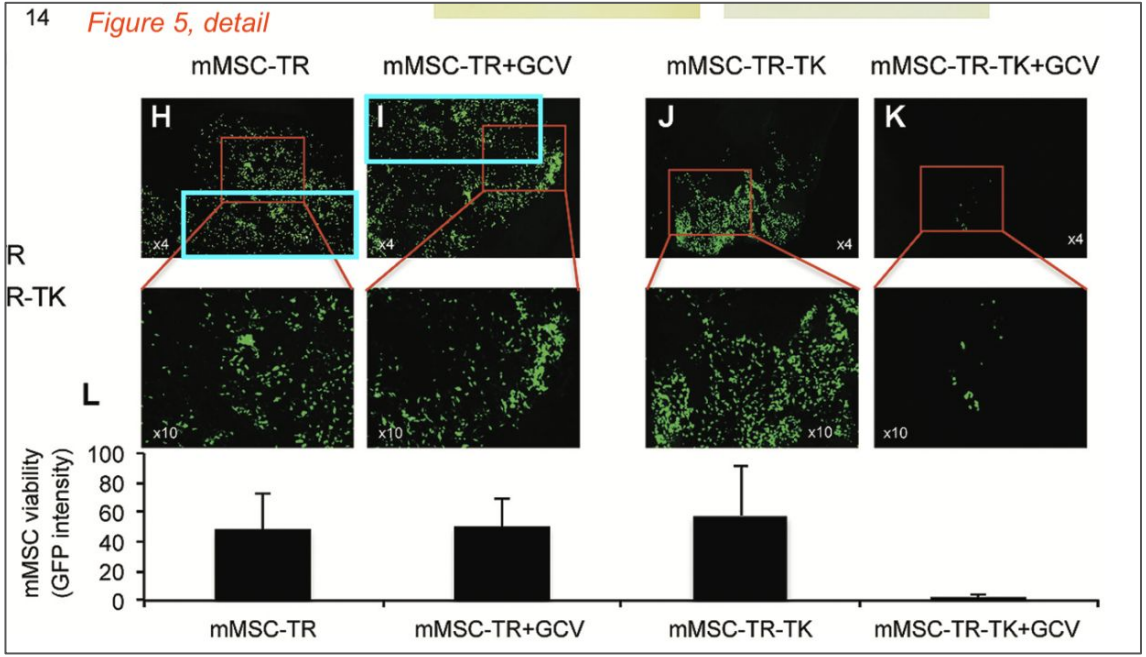
Therapeutic Efficacy and Fate of Bimodal Engineered Stem Cells in Malignant Brain Tumors

JORDI MARTINEZ-QUINTANILLA,[a,b] DEEPAK BHERE,[a,b] PEDRAM HEIDARI,[b,c] DEREK HE,[a,b] UMAR MAHMOOD,[b,c] KHALID SHAH[a,b,d,e]

[a]Molecular Neurotherapy and Imaging Laboratory, [b]Department of Radiology, [c]Division of Nuclear Medicine and Molecular Imaging, and [d]Department of Neurology, Massachusetts General Hospital, Harvard Medical School, Boston, Massachusetts, USA, [e]Harvard Stem Cell Institute, Harvard University, Cambridge, Massachusetts, USA

2013

Figure 5:
Cyan boxes highlight an overlap observed between panels H and I, which are labeled differently.



Therapeutics, Targets, and Chemical Biology

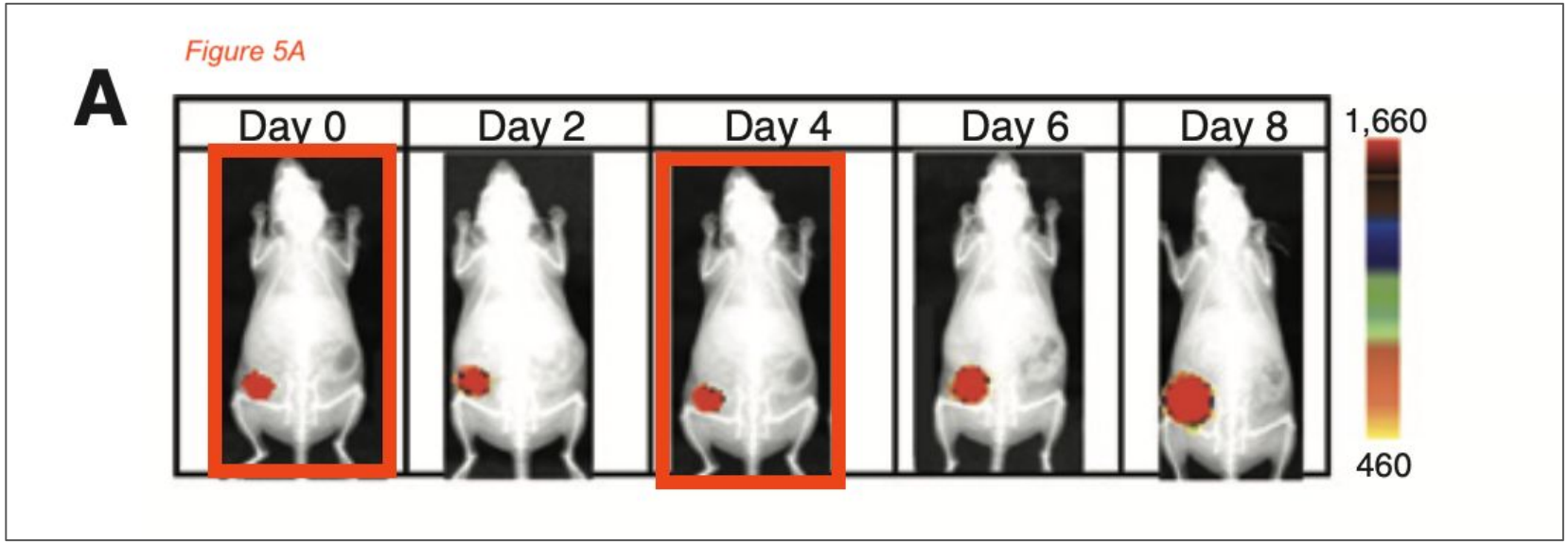
Cancer Research

Molecular Imaging with Bioluminescence and PET Reveals Viral Oncolysis Kinetics and Tumor Viability

Darshini Kuruppu[1], Anna-Liisa Brownell[2], Khalid Shah[2], Umar Mahmood[2], and Kenneth K. Tanabe[1]

2014

Figure 5A:
Red boxes highlight that the Day 0 and Day 4 mice look unexpectedly similar to each other. The bioluminescence signal is slightly different, but positions of gut air bubble and legs look very similar.



Therapeutics, Targets, and Chemical Biology

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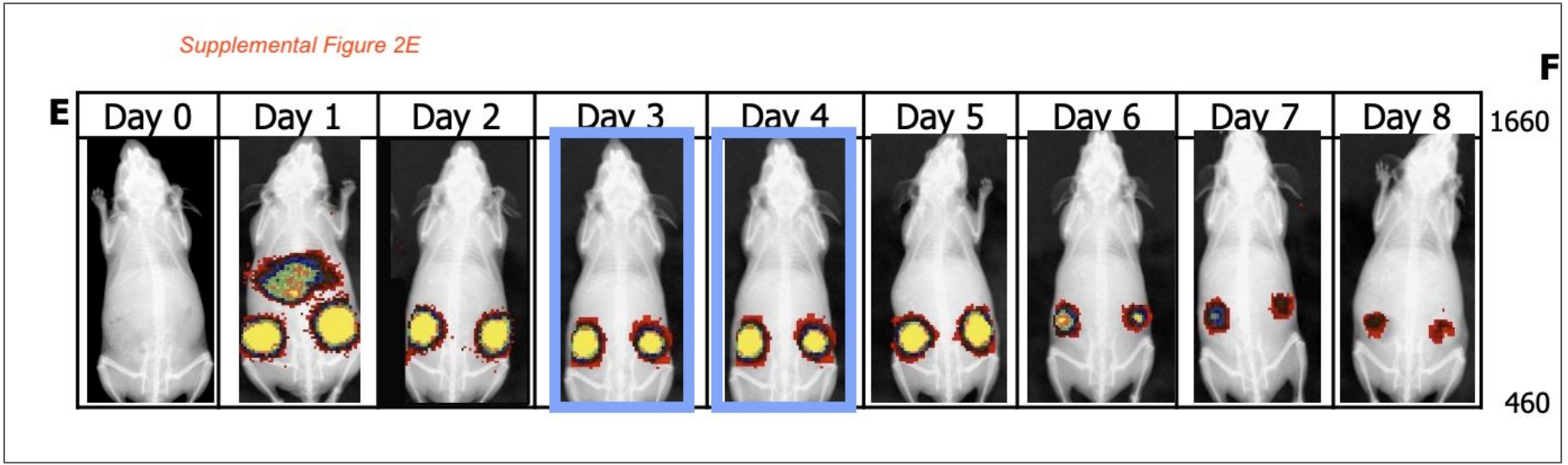
Darshini Kuruppu[1], Anna-Liisa Brownell[2], Khalid Shah[2], Umar Mahmood[2], and Kenneth K. Tanabe[1]

2014

Supplemental Figure 2E:
Blue boxes highlight that the Day 3 and Day 4 mice look unexpectedly similar to each other.
The bioluminescence signal also looks the same.
The scale bar on the right is missing.



original article

Molecular Therapy (2015), DOI: 10.1038/mt.2014.214

Antiangiogenic Variant of TSP-1 Targets Tumor Cells in Glioblastomas

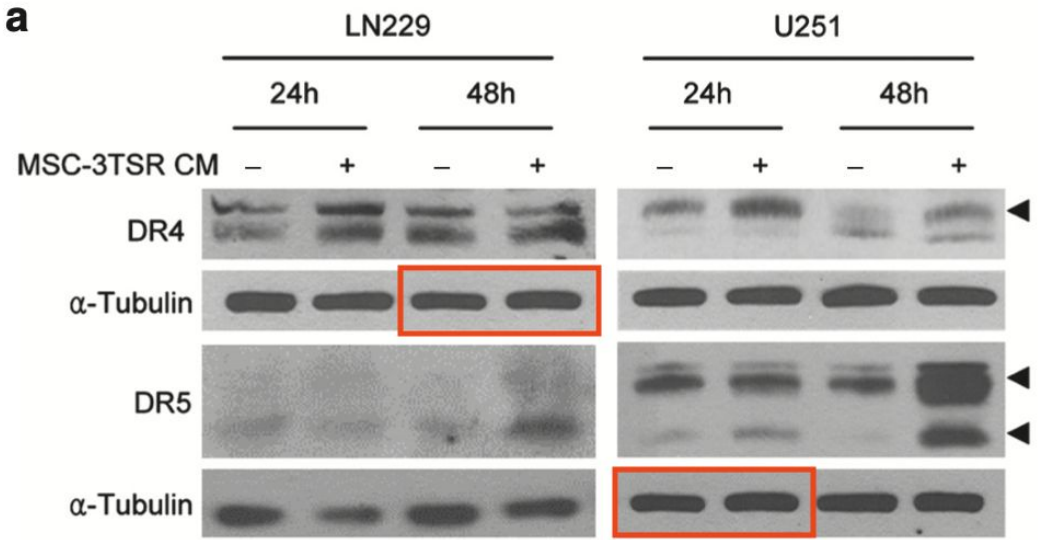
Sung Hugh Choi[1], Kaoru Tamura[1], Rajiv Kumar Khajuria[1], Deepak Bhere[1], Irina Nesterenko[1], Jack Lawler[2] and Khalid Shah[1,3,4]

[1]Molecular Neurotherapy and Imaging Laboratory, Department of Radiology, Massachusetts General Hospital, Harvard Medical School, Boston, Massachusetts, USA; [2]Division of Experimental Pathology, Department of Pathology, Beth Israel Deaconess Medical Center and Harvard Medical School, Boston, Massachusetts, USA; [3]Department of Neurology, Massachusetts General Hospital, Harvard Medical School, Boston, Massachusetts, USA; [4]Harvard Stem Cell Institute, Harvard University, Cambridge, Massachusetts, USA

2015

Red boxes:
Two lanes in the LN229 tubulin blot appear to overlap with two lanes in the U251 tubulin blot. The two lanes represent different time points and cell lines.

Figure 2a



original article

Molecular Therapy (2015), DOI: 10.1038/mt.2014.214

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Sung Hugh Choi[1], Kaoru Tamura[1], Rajiv Kumar Khajuria[1], Deepak Bhere[1], Irina Nesterenko[1], Jack Lawler[2] and Khalid Shah[1,3,4]

[1]Molecular Neurotherapy and Imaging Laboratory, Department of Radiology, Massachusetts General Hospital, Harvard Medical School, Boston, Massachusetts, USA; [2]Division of Experimental Pathology, Department of Pathology, Beth Israel Deaconess Medical Center and Harvard Medical School, Boston, Massachusetts, USA; [3]Department of Neurology, Massachusetts General Hospital, Harvard Medical School, Boston, Massachusetts, USA; [4]Harvard Stem Cell Institute, Harvard University, Cambridge, Massachusetts, USA

2015

Pale-red boxes:
The LN229 tubulin blot in Figure 2a (established glioblastoma cell lines) looks the same as the tubulin blot in Figure 3C (human brain microvascular endothelial cells, HBMVEC).

Figure 2a: established glioblastoma cell lines

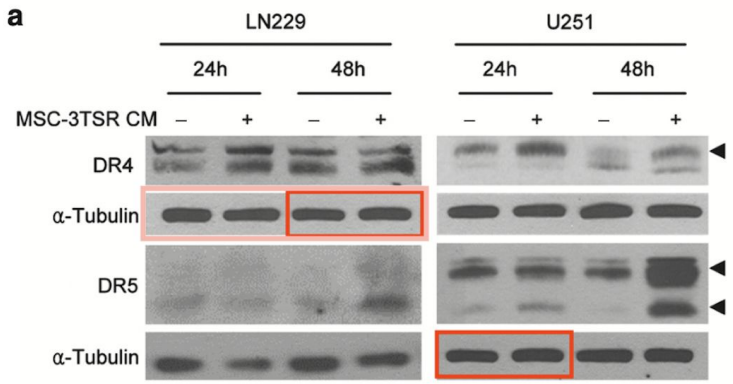
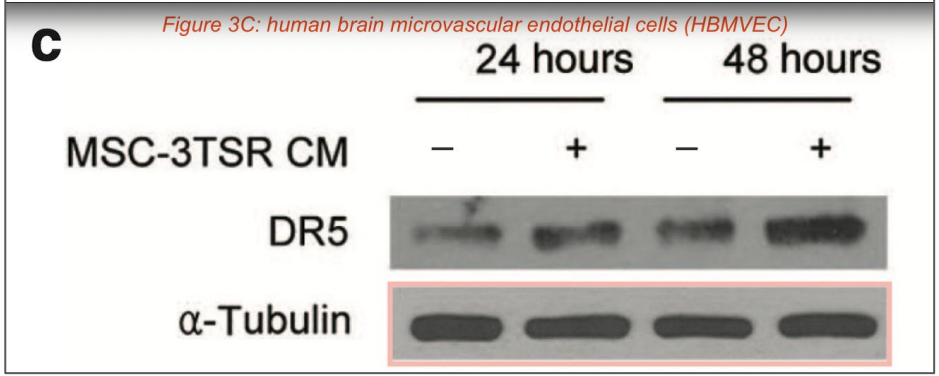


Figure 3C: human brain microvascular endothelial cells (HBMVEC)

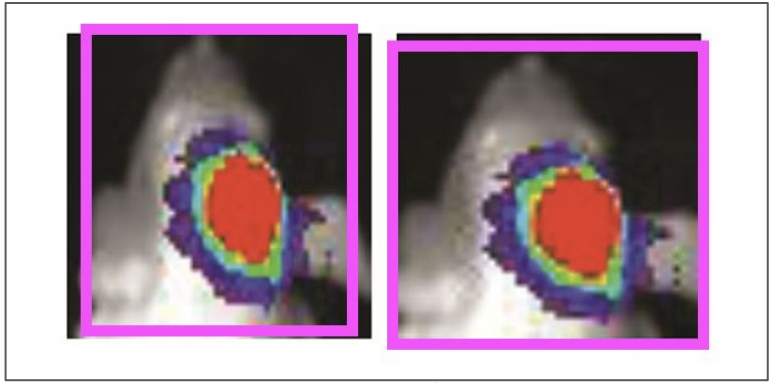


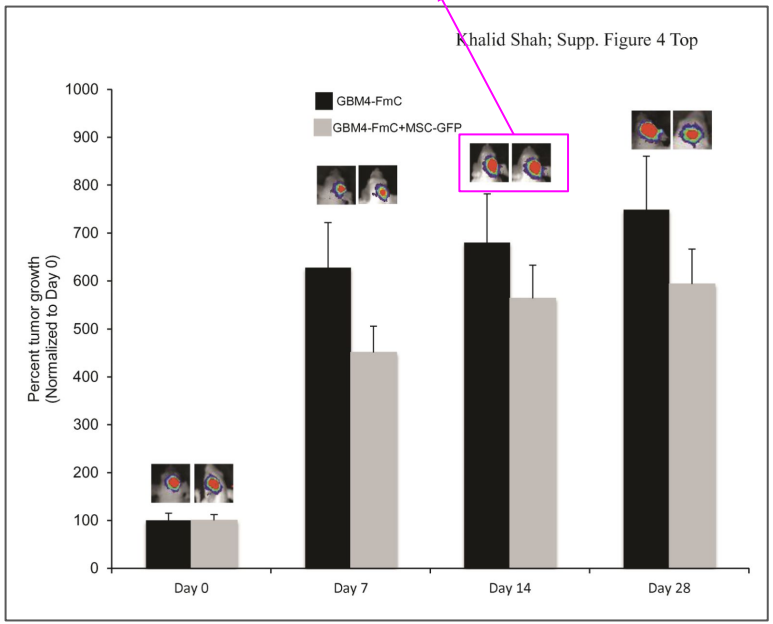
STEM CELLS
TRANSLATIONAL AND CLINICAL RESEARCH

Combination of Systemic Chemotherapy with Local Stem Cell Delivered S-TRAIL in Resected Brain Tumors

NAVID REDJAL,[a,b,c] YANNI ZHU,[a,b] KHALID SHAH[a,b,d,e]

2015





Supplemental Figure 4.
Pink boxes:
The two mouse luminescence photos above the Day 14 bars appear to show the same animal, albeit stretched differently.
The top photo shows the two images in more detail.
The photos are representing differently treated animals, i.e., GBM4-Fmc vs. GBM40FmC+MSC-GFP, respectively.

Cancer Therapy: Preclinical

Clinical Cancer Research

Tumor Resection Recruits Effector T Cells and Boosts Therapeutic Efficacy of Encapsulated Stem Cells Expressing IFNβ in Glioblastomas

Sung Hugh Choi[1,2], Daniel W. Stuckey[1], Sara Pignatta[3], Clemens Reinshagen[1,2], Jasneet Kaur Khalsa[1,2], Nicolaas Roozendaal[1], Jordi Martinez-Quintanilla[1], Kaoru Tamura[1], Erhan Keles[1], and Khalid Shah[1,2,4]

ImageTwin found that the +gel panels in Figure 3A look identical to the PBS panels in Figure 3F.
The labels suggest these are different experiments. Are they?

2017

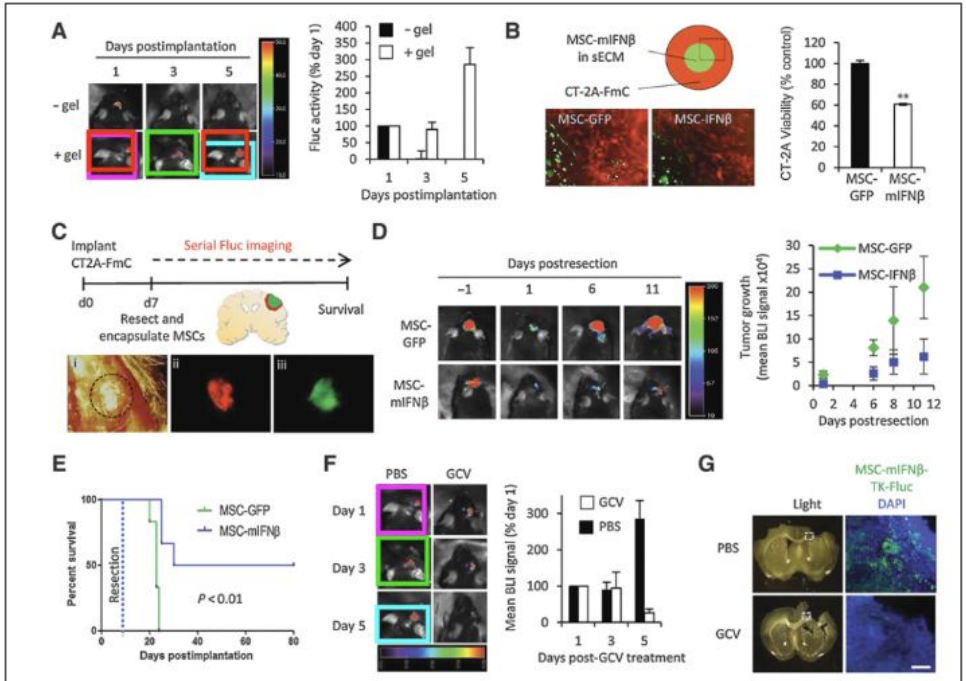


Figure 3.
MSC-mIFNβ show antitumor efficacy in resected GBM, leading to increased survival of mice and can be eliminated posttherapy. **A,** Representative BLIs and plot of mean Fluc signal intensity of mice bearing intracranial MSC-GFP-Fluc cells with or without sECM encapsulation (1×10^6 cells/mouse, $n = 4$/group). **B,** Schematic and fluorescence images showing MSC-GFP or MSC-mIFNβ cells encapsulated in sECM (1×10^4 cells/drop), surrounded by CT2A-FmC cells. Plot showing CT2A cell viability at day5. **C,** Scheme for testing efficacy of sECM-encapsulated MSCs expressing GFP or mIFNβ on resected CT2A-FmC tumors ($n = 6$/group): (i) light image of a craniotomy above tumor, delineated by dashed black circle; (ii, iii) fluorescence images of before-resected CT2A-FmC tumor (red) and encapsulated MSC-mIFNβ cells in the resection cavity (green). **D** and **E,** Representative BLIs of mice from MSC-GFP and -mIFNβ groups pre- and postresection (**D**), plot of mean tumor growth and survival curves (**E**). **F** and **G,** Resected mice were implanted with sECM-encapsulated MSC-mIFNβ-TK-Fluc ($n = 6$) and treated with GCV (50 mg/kg) or PBS daily for 5 days. **F,** Representative BLIs of mice at 1, 3, and 5 days after GCV treatment and plot of mean tumor growth. **G,** Light and fluorescence micrographs of coronal brain sections containing encapsulated MSC-mIFNβ-TK-Fluc (green) from brains harvested 7 days after GCV treatment and DAPI counterstained (blue). White dashed box indicates region of interest. Scale bar, 100 μm.

†KS and HW are the co-senior authors of this work.

Grant sponsor: This work was supported by JSMF (KS) and ABTA (HW) and NIH-R01CA204720 (KS)

DOI: 10.1002/ijc.30811

IJC
International Journal of Cancer

Therapeutic targeting of chemoresistant and recurrent glioblastoma stem cells with a proapoptotic variant of oncolytic herpes simplex virus

Nusrat Jahan[1,2], Jae M. Lee[1,2], Khalid Shah[1,2,3,4†] and Hiroaki Wakimoto[1,2,5†]

[1] Department of Radiology, Center for Stem Cell Therapeutics and Imaging, Massachusetts General Hospital, Harvard Medical School, Boston, MA
[2] Department of Radiology, Massachusetts General Hospital, Harvard Medical School, Boston, MA
[3] Department of Neurology, Massachusetts General Hospital, Harvard Medical School, Boston, MA
[4] Harvard Stem Cell Institute, Harvard University, Cambridge, MA
[5] Department of Neurosurgery, Massachusetts General Hospital, Harvard Medical School, Boston, MA

The Control (PBS) panels in Figures 4g (GSC23mC implant) and 5d (GSC31 implant) overlap. The experimental schemas in Figures 4d and 5a suggest these are different experiments.

2017

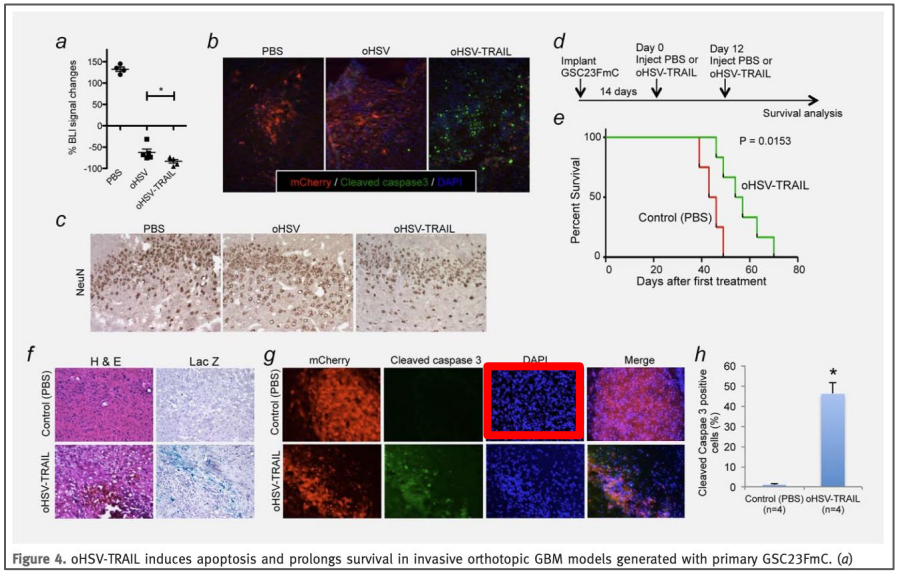


Figure 4. oHSV-TRAIL induces apoptosis and prolongs survival in invasive orthotopic GBM models generated with primary GSC23FmC. (*a*)

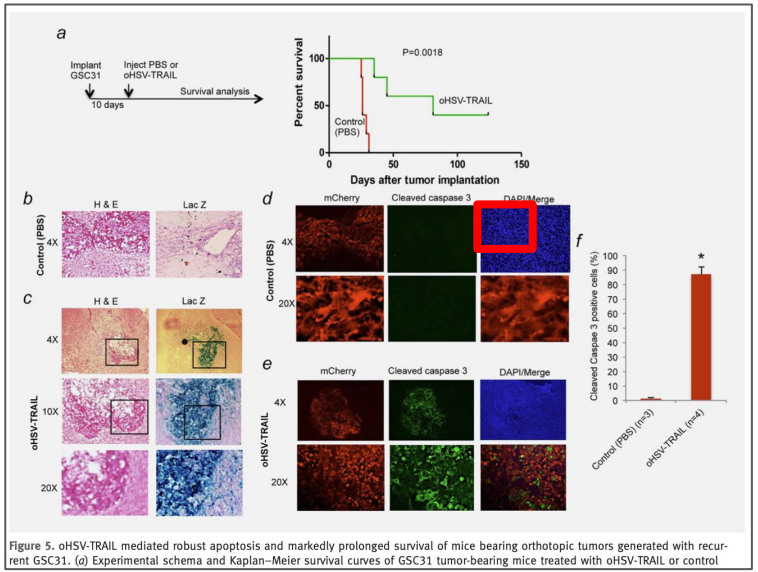


Figure 5. oHSV-TRAIL mediated robust apoptosis and markedly prolonged survival of mice bearing orthotopic tumors generated with recurrent GSC31. (*a*) Experimental schema and Kaplan–Meier survival curves of GSC31 tumor-bearing mice treated with oHSV-TRAIL or control

†KS and HW are the co-senior authors of this work.

Grant sponsor: This work was supported by JSMF (KS) and ABTA (HW) and NIH-R01CA204720 (KS)

DOI: 10.1002/ijc.30811

IJC
International Journal of Cancer

Therapeutic targeting of chemoresistant and recurrent glioblastoma stem cells with a proapoptotic variant of oncolytic herpes simplex virus

Nusrat Jahan[1,2], Jae M. Lee[1,2], Khalid Shah [1,2,3,4†] and Hiroaki Wakimoto[1,2,5†]

[1] Department of Radiology, Center for Stem Cell Therapeutics and Imaging, Massachusetts General Hospital, Harvard Medical School, Boston, MA
[2] Department of Radiology, Massachusetts General Hospital, Harvard Medical School, Boston, MA
[3] Department of Neurology, Massachusetts General Hospital, Harvard Medical School, Boston, MA
[4] Harvard Stem Cell Institute, Harvard University, Cambridge, MA
[5] Department of Neurosurgery, Massachusetts General Hospital, Harvard Medical School, Boston, MA

Close up view of the problem noted on the previous slide. Also note that the mCherry signal is different between both figures and that the Merge image in Figure 4g does not match the mCherry red signal.

2017

Figure 4g

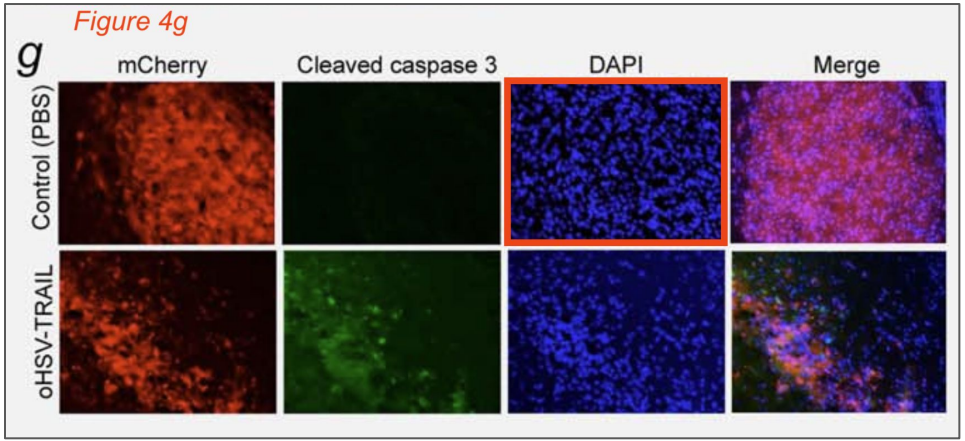
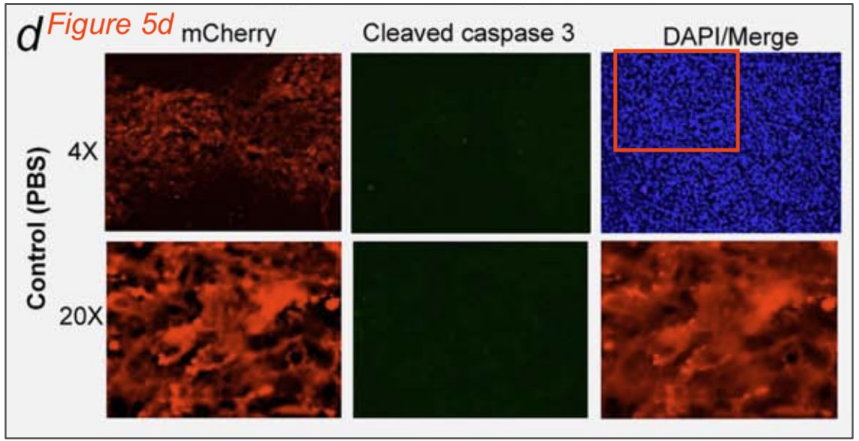


Figure 5d



2017

†KS and HW are the co-senior authors of this work.

Grant sponsor: This work was supported by JSMF (KS) and ABTA (HW) and NIH-R01CA204720 (KS)

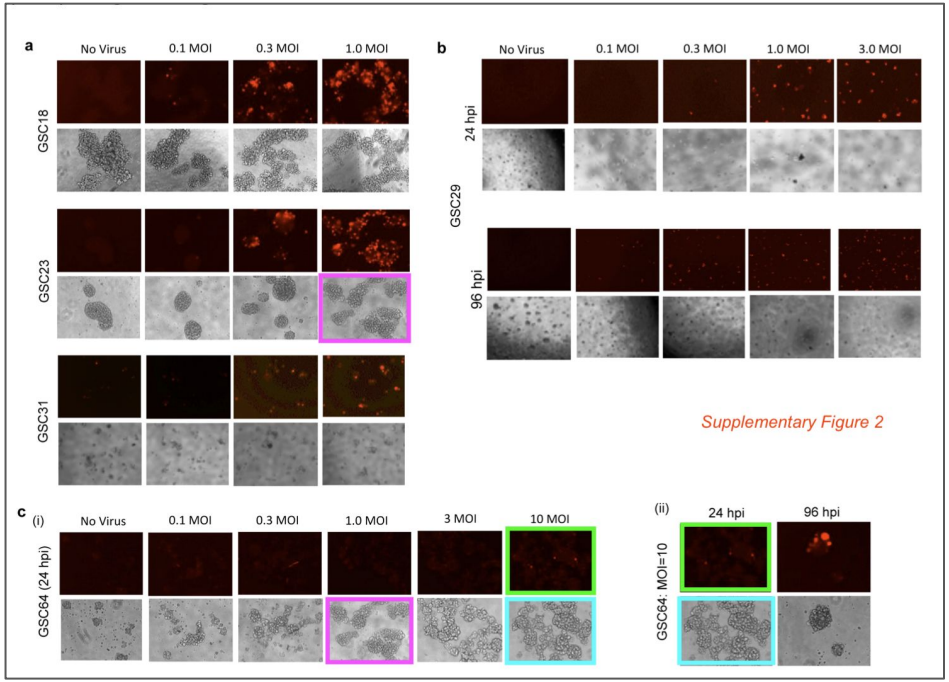
DOI: 10.1002/ijc.30811

IJC
International Journal of Cancer

Therapeutic targeting of chemoresistant and recurrent glioblastoma stem cells with a proapoptotic variant of oncolytic herpes simplex virus

Nusrat Jahan[1,2], Jae M. Lee[1,2], Khalid Shah[1,2,3,4†] and Hiroaki Wakimoto[1,2,5†]

[1] Department of Radiology, Center for Stem Cell Therapeutics and Imaging, Massachusetts General Hospital, Harvard Medical School, Boston, MA
[2] Department of Radiology, Massachusetts General Hospital, Harvard Medical School, Boston, MA
[3] Department of Neurology, Massachusetts General Hospital, Harvard Medical School, Boston, MA
[4] Harvard Stem Cell Institute, Harvard University, Cambridge, MA
[5] Department of Neurosurgery, Massachusetts General Hospital, Harvard Medical School, Boston, MA



Supplementary Figure 2:
** Pink boxes: The grey GSC23 1.0 MOI and the GSC64 1.0 MOI panels look unexpectedly identical. The mCherry red panels look different (as expected).*
** Cyan and green boxes highlight an expected and appropriate set of identical panels (these are not a problem).*

Green boxes:
Panels in Figure 4E and 5F of this paper appear to show the same tissue sample, albeit 90 degree rotated and at different magnifications. The labels suggest these are different experiments.

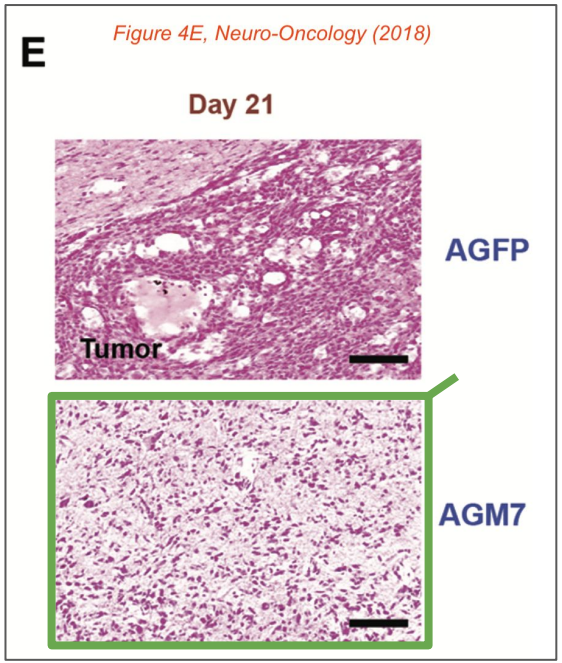
Neuro-Oncology
20(2), 215–224, 2018 | doi:10.1093/neuonc/nox138 | Advance Access date 25 July 2017

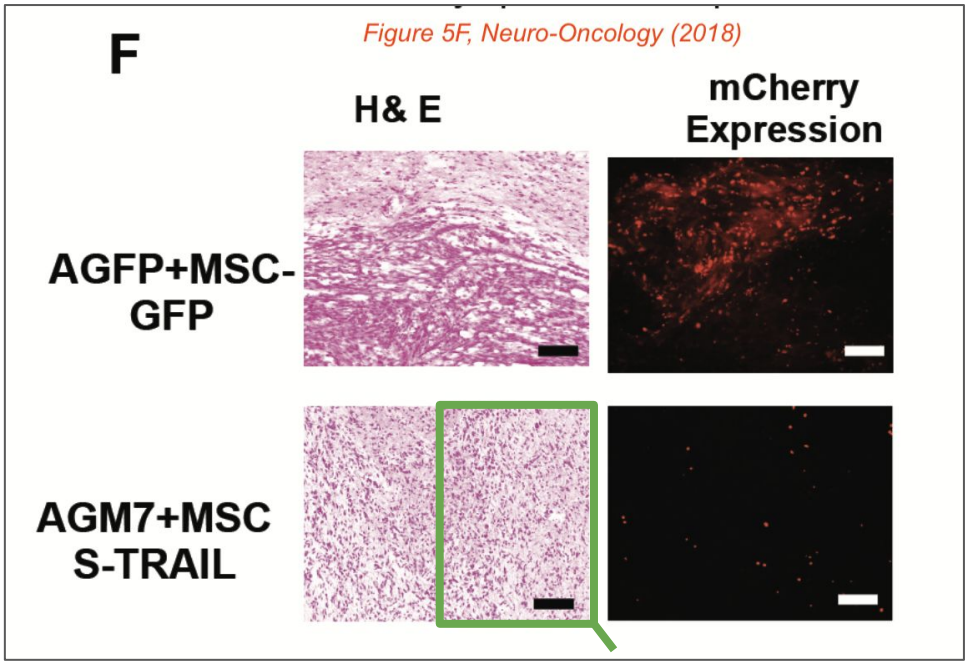
microRNA-7 upregulates death receptor 5 and primes resistant brain tumors to caspase-mediated apoptosis

Deepak Bhere,* Kaoru Tamura,* Hiroaki Wakimoto, Sung Hugh Choi, Benjamin Purow, Jeremy Debatisse, and Khalid Shah

Center for Stem Cell Therapeutics and Imaging (D.B., K.T, H.W., S.H.C., J.D., K.S.), Department of Radiology (D.B., K.T., H.W., S.H.C., J.D., K.S.), Department of Neurosurgery (H.W.), and Department of Neurology, Massachusetts General Hospital, Harvard Medical School, Boston, Massachusetts (K.S.); Department of Neurology, University of Virginia, Charlottesville, Virginia (B.P.); Harvard Stem Cell Institute, Harvard University, Cambridge, Massachusetts (K.S.); Center for Stem Cell Therapeutics and Imaging, Department of Neurosurgery, Brigham and Women's Hospital, Harvard Medical School, Boston, Massachusetts (D.B., H.W., S.H.C., K.S.)

2018





SCIENTIFIC REPORTS

OPEN

2017

Received: 31 January 2017
Accepted: 11 April 2017
Published online: 01 June 2017

Bi-specific molecule against EGFR and death receptors simultaneously targets proliferation and death pathways in tumors

Yanni Zhu[1,3], Nicole Bassoff[1,3], Clemens Reinshagen[1,2,3,5], Deepak Bhere[1,2,3,5], Michal O. Nowicki[5], Sean E. Lawler[5], Jérémie Roux[6] & Khalid Shah[1,2,3,4,5,7]

Neuro-Oncology

20(2), 215–224, 2018 | doi:10.1093/neuonc/nox138 | Advance Access date 25 July 2017

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2018

2017 Figure 1C

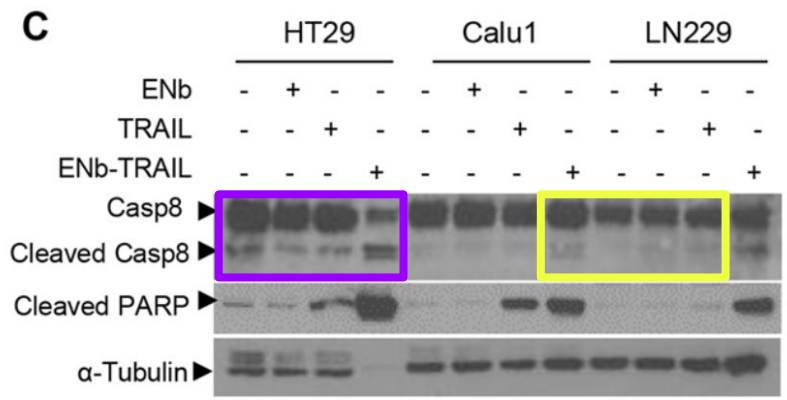
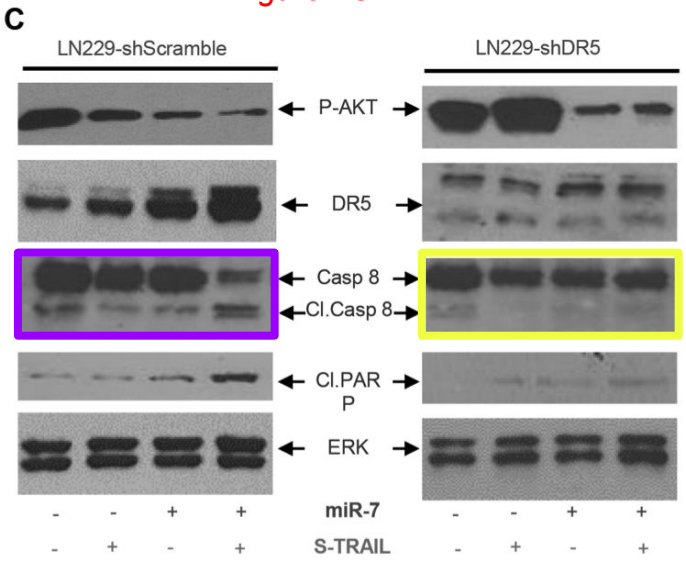


Figure 2C



Purple and yellow boxes highlight blots used in two different papers, representing different cell lines and experiments.

SCIENTIFIC REPORTS

OPEN

2017

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microRNA-7 upregulates death receptor 5 and primes resistant brain tumors to caspase-mediated apoptosis

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Center for Stem Cell Therapeutics and Imaging (D.B., K.T, H.W., S.H.C., J.D., K.S.), Department of Radiology (D.B., K.T, H.W., S.H.C., J.D., K.S.), Department of Neurosurgery (H.W.), and Department of Neurology, Massachusetts General Hospital, Harvard Medical School, Boston, Massachusetts (K.S.); Department of Neurology, University of Virginia, Charlottesville, Virginia (B.P.); Harvard Stem Cell Institute, Harvard University, Cambridge, Massachusetts (K.S.); Center for Stem Cell Therapeutics and Imaging, Department of Neurosurgery, Brigham and Women's Hospital, Harvard Medical School, Boston, Massachusetts (D.B., H.W., S.H.C., K.S.)

2018

Blue and red boxes highlight figure panels used in two different papers from the same research group, but the different labels suggest these were different experiments.

Figure 5C, Scientific Reports (2017)

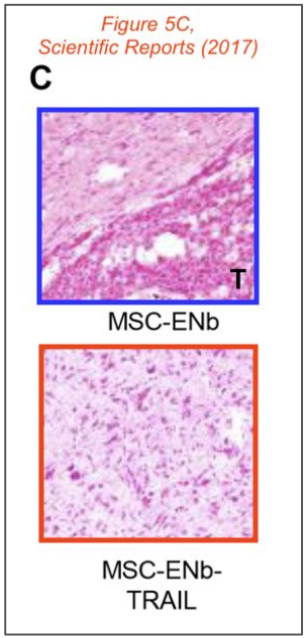
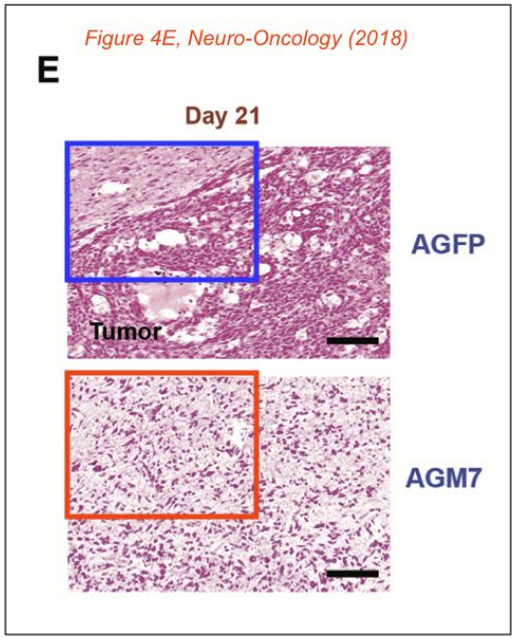


Figure 4E, Neuro-Oncology (2018)



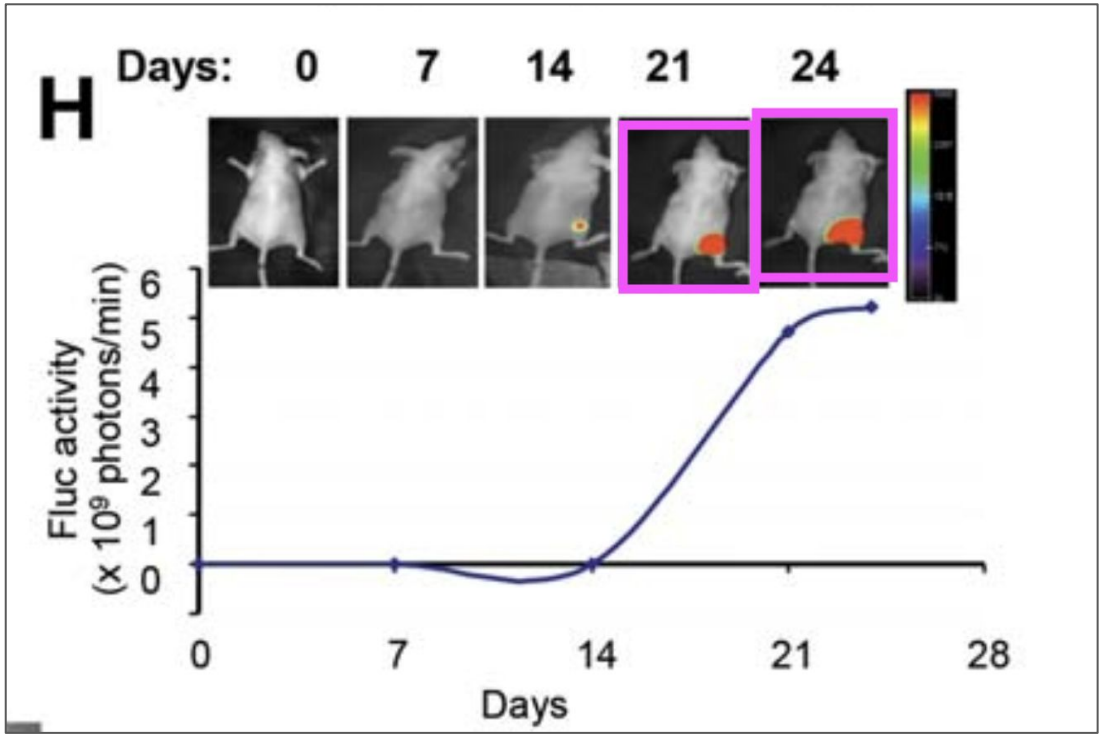
STEM CELLS
TRANSLATIONAL AND CLINICAL RESEARCH

Stem Cells Engineered During Different Stages of Reprogramming Reveal Varying Therapeutic Efficacies

DEEPAK BHERE,[a,b,g,h*] RAJIV KUMAR KHAJURIA,[a,b*] WILLIAM T. HENDRIKS,[c,d,e] ANTARA BANDYOPADHYAY,[a,b,g,h] TUGBA BAGCI-ONDER,[a,b] KHALID SHAH [a,b,e,f,h]

2018

Figure 1H:
Pink boxes: The photos of the mice at 21 and 24 days look more similar than expected, although the luminescence patterns differ.



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2018

Figure 2A and B:
Red boxes: In the bottom row, three of the six panels appear to be showing the same photo, i.e. the one belonging to the EB panel. Image made lighter to bring out the background.

Figure 2A and 2B

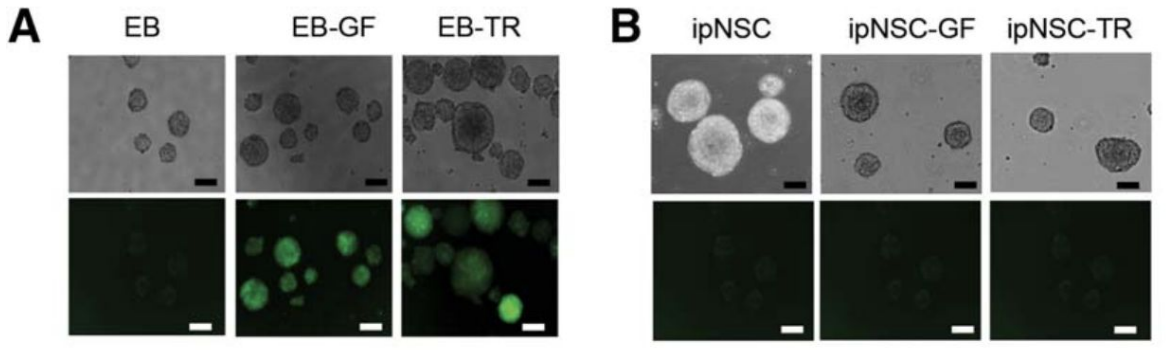
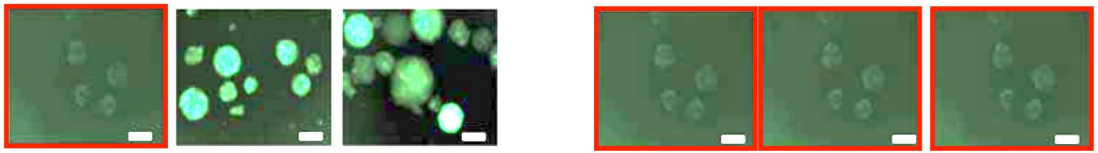


Figure 2A and 2B, bottom row, background enhanced



STEM CELLS
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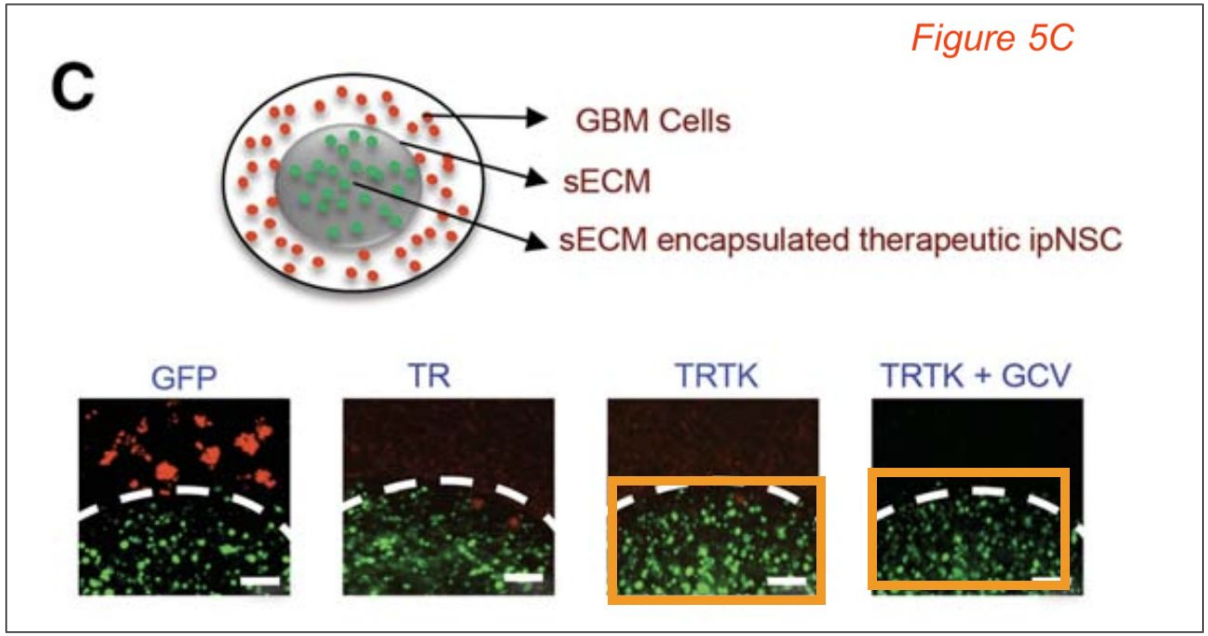
2018

Figure 5C:
Orange boxes: The TRTK and TRTK+GCV panels appear to show the same specimen.



Cancer Gene Therapy (2019) 26:145–156
https://doi.org/10.1038/s41417-018-0060-z

ARTICLE

A model of breast cancer meningeal metastases: characterization with in vivo molecular imaging

Darshini Kuruppu[1] · Deepak Bhere[2] · Christian T. Farrar[3] · Khalid Shah[2] · Anna-Liisa Brownell[3] · Kenneth K. Tanabe[1]

2019

Figure 3:
** Red boxes: one of the Sham Brain MRI photos seems identical to a Day 9 Tumor Brain MRI photo, albeit stretched differently.*
** Yellow, pink, and cyan boxes: Three sets of Day 19 and Day 21 MRI photos look unexpectedly similar.*

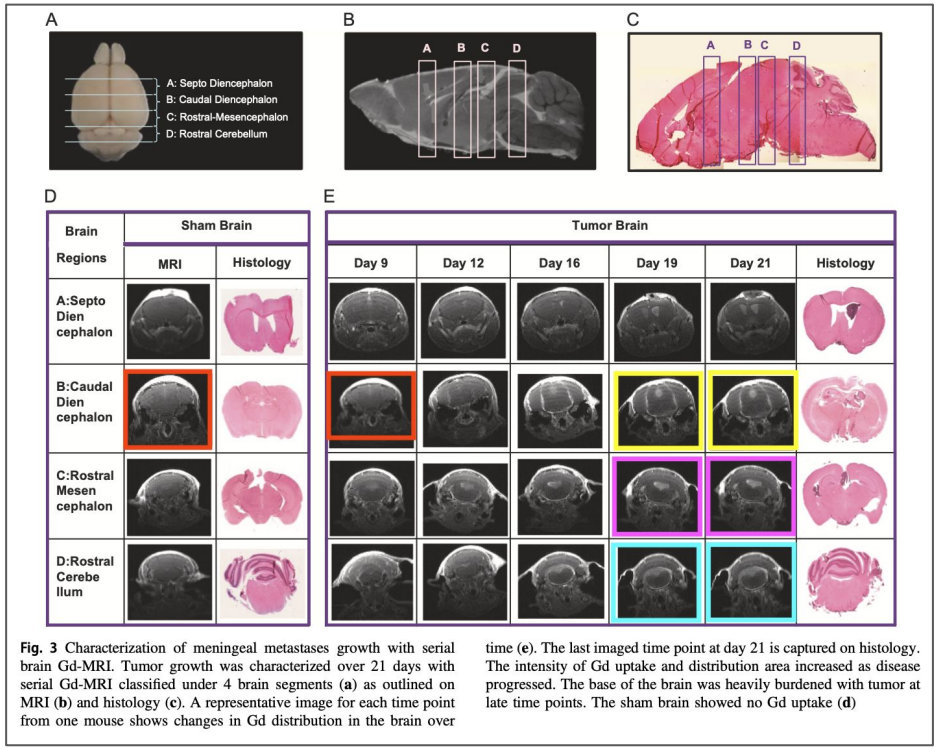


Fig. 3 Characterization of meningeal metastases growth with serial brain Gd-MRI. Tumor growth was characterized over 21 days with serial Gd-MRI classified under 4 brain segments (**a**) as outlined on MRI (**b**) and histology (**c**). A representative image for each time point from one mouse shows changes in Gd distribution in the brain over time (**e**). The last imaged time point at day 21 is captured on histology. The intensity of Gd uptake and distribution area increased as disease progressed. The base of the brain was heavily burdened with tumor at late time points. The sham brain showed no Gd uptake (**d**)

2020

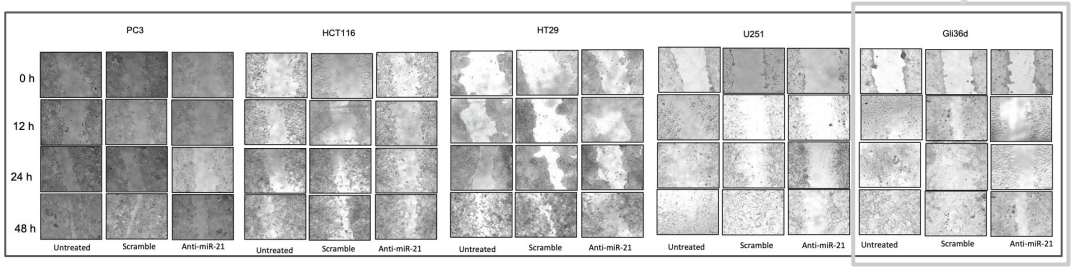
SCIENTIFIC REPORTS
nature research

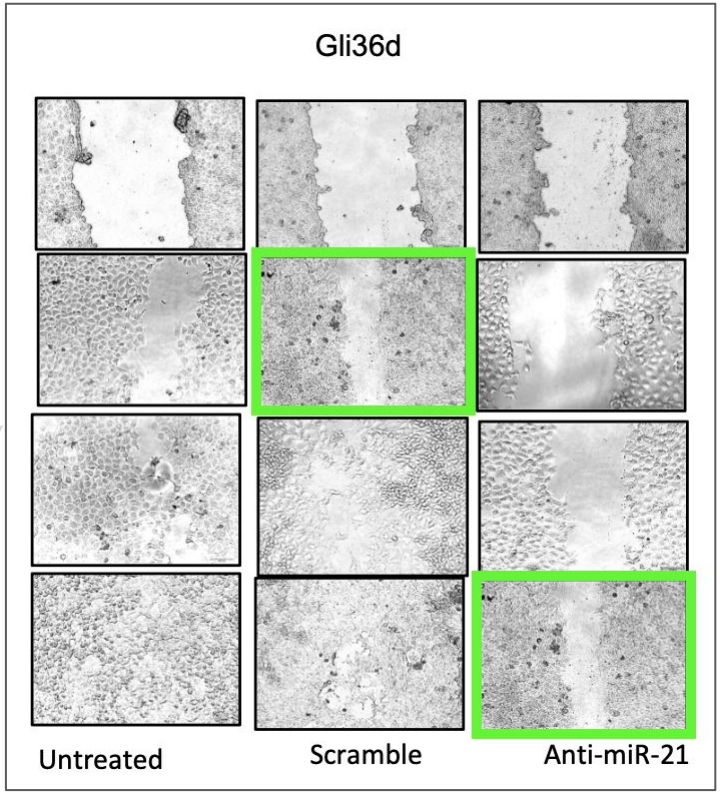
Simultaneous downregulation of miR-21 and upregulation of miR-7 has anti-tumor efficacy

Deepak Bhere[1,2,6], Nahid Arghiani[1,2,3,6], Esther Revai Lechtich[1,2], Yizheng Yao[2], Sarah Alsaab[1,2,4], Fengfeng Bei[2], Maryam M. Matin[3] & Khalid Shah[1,2,5*]

SCIENTIFIC REPORTS | (2020) 10:1779 | https://doi.org/10.1038/s41598-020-58072-w

Supplemental Figure 4:
** Green boxes: One of the Gli36d/Scramble panels looks the same as a Gli36d/Anti-miR-21 panel.*





2020

nature COMMUNICATIONS

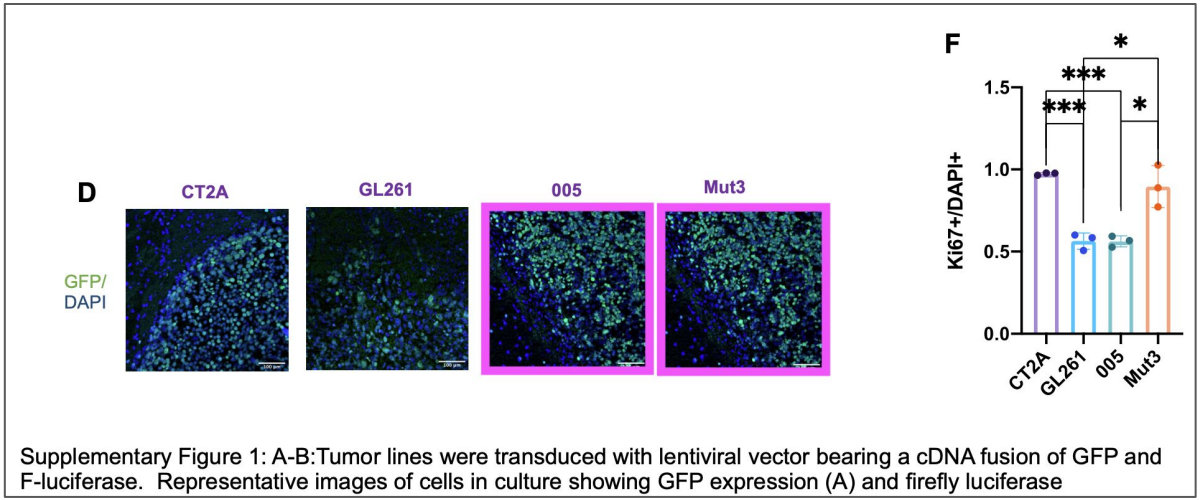
ARTICLE

https://doi.org/10.1038/s41467-020-17704-5 OPEN

Immune phenotyping of diverse syngeneic murine brain tumors identifies immunologically distinct types

Jasneet Kaur Khalsa[1,2], Nina Cheng[1,2], Joshua Keegan[3], Ameen Chaudry[1], Joseph Driver[2], Wenya Linda Bi[2], James Lederer[3] & Khalid Shah[1,2,4]✉

Supplemental Figure 1D:
** Pink boxes:*
The 005 and Mut3 panels look identical, while they are presented as differently transduced cell lines.
In addition, the data in Figure 1F show significantly different measurements.



Supplementary Figure 1: A-B:Tumor lines were transduced with lentiviral vector bearing a cDNA fusion of GFP and F-luciferase. Representative images of cells in culture showing GFP expression (A) and firefly luciferase

Oncogene (2021) 40:3201–3216
https://doi.org/10.1038/s41388-021-01697-6

ARTICLE

Generation of TRAIL-resistant cell line models reveals distinct adaptive mechanisms for acquired resistance and re-sensitization

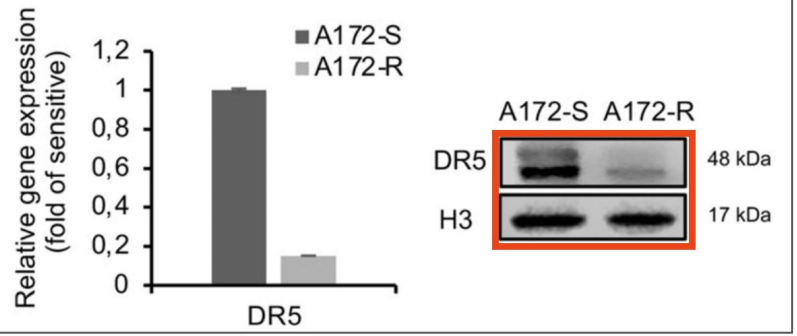
Ahmet Cingöz[1,2] · Ezgi Ozyerli-Goknar[1,2] · Tunc Morova[2] · Fidan Seker-Polat[1,2] · Myvizhi Esai Selvan[3,4] · Zeynep Hülya Gümüş[2,3,4] · Deepak Bhere[5] · Khalid Shah[5] · Ihsan Solaroglu[2,6] · Tugba Bagci-Onder[1,2]

Received: 13 May 2019 / Revised: 21 January 2021 / Accepted: 4 February 2021 / Published online: 25 March 2021

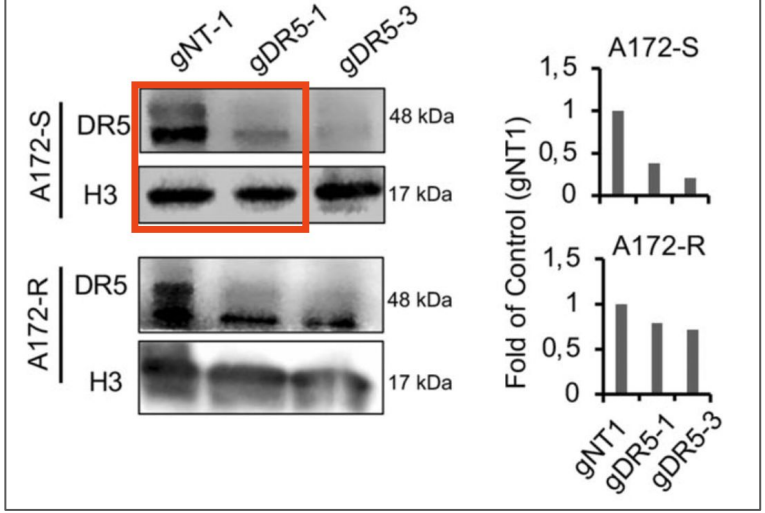
2021

Figures 4A and 4F:
Red boxes: For both the DR5 and H3 blots, the A172-S and A172-R lanes in Figure 4A look identical to the A172-S gNT-1 and gDR5-1 lanes in Figure 4F.

A *Figure 4A*



F *Figure 4F*



Oncogene (2021) 40:3201–3216
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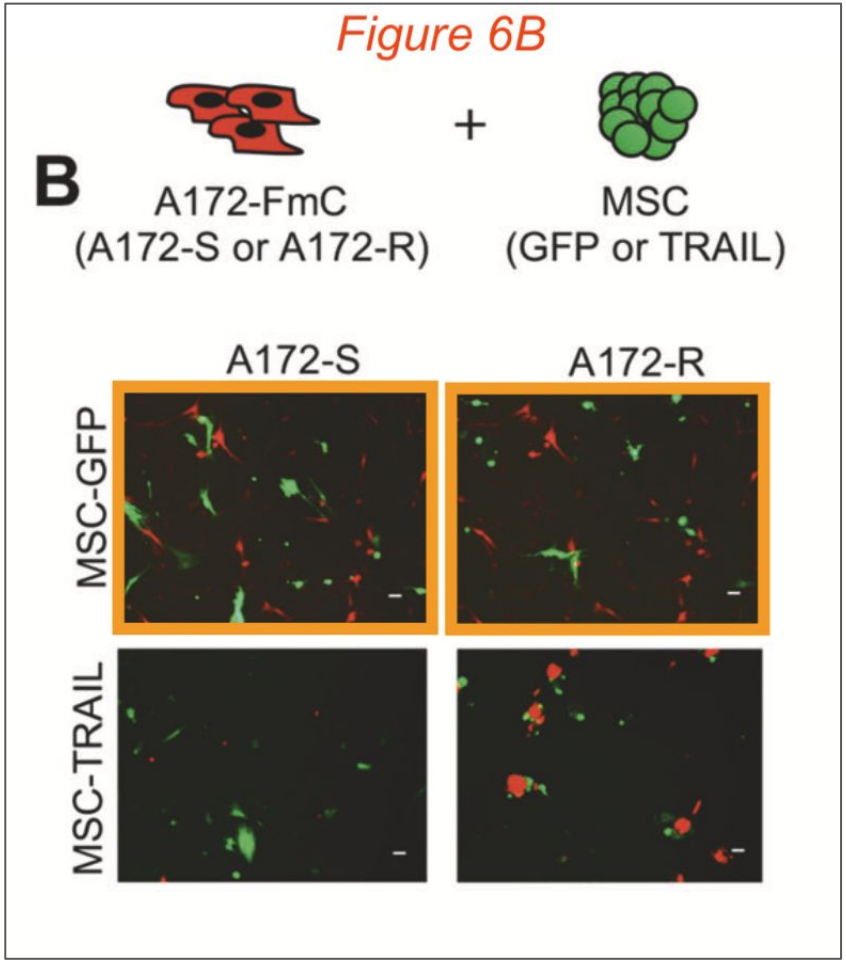
Received: 13 May 2019 / Revised: 21 January 2021 / Accepted: 4 February 2021 / Published online: 25 March 2021

Figure 6B:
Orange boxes: The red signals in the MSC-GFP/A172-S and MSC-GFP/A172-R panels look identical. The green signals are different.



2021

Oncogene (2021) 40:3201–3216
https://doi.org/10.1038/s41388-021-01697-6

ARTICLE

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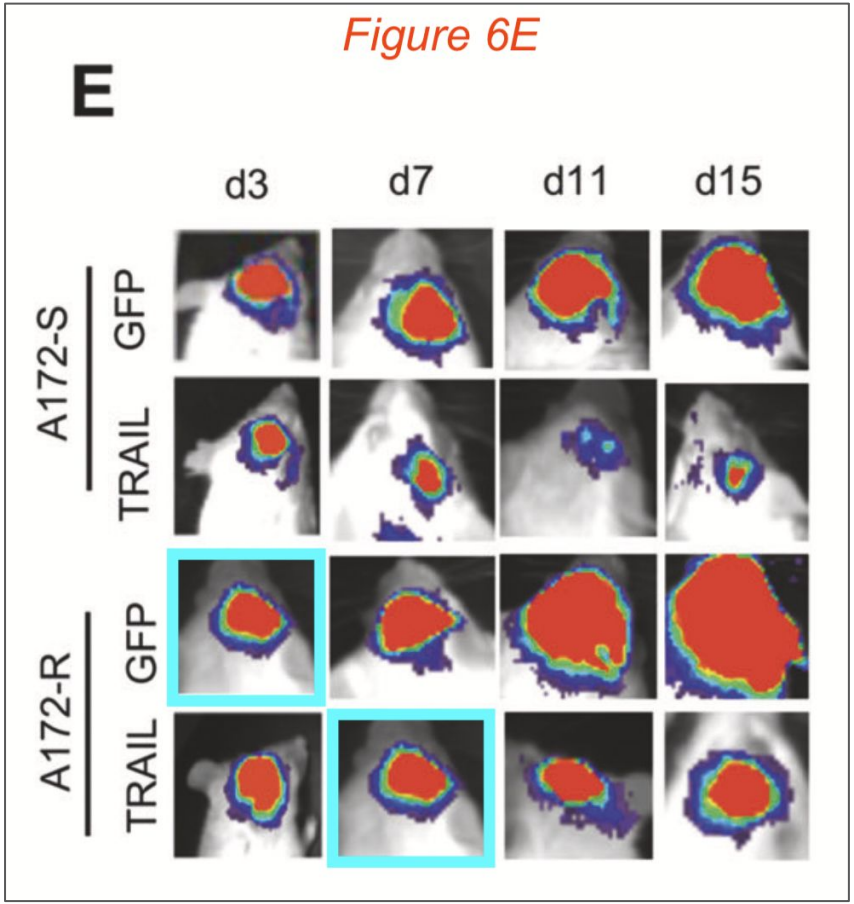
Received: 13 May 2019 / Revised: 21 January 2021 / Accepted: 4 February 2021 / Published online: 25 March 2021

Figure 6E:
Cyan boxes: The d3/A172-R/GFP and the d7/A172-R/TRAIL panels look identical.



2021

OPEN ACCESS Freely available online

PLOS ONE (2010)

PLoS one

Analysis of Death Receptor 5 and Caspase-8 Expression in Primary and Metastatic Head and Neck Squamous Cell Carcinoma and Their Prognostic Impact

Heath A. Elrod[1], Songqing Fan[1], Susan Muller[2], Georgia Z. Chen[1], Lin Pan[3], Mourad Tighiouart[3], Dong M. Shin[1], Fadlo R. Khuri[1], Shi-Yong Sun[1]*

1 Department of Hematology and Medical Oncology, Emory University School of Medicine and Winship Cancer Institute, Atlanta, Georgia, United States of America, **2** Department of Pathology, Emory University School of Medicine and Winship Cancer Institute, Atlanta, Georgia, United States of America, **3** Department of Biostatistics and Bioinformatics, Emory University School of Medicine and Winship Cancer Institute, Atlanta, Georgia, United States of America

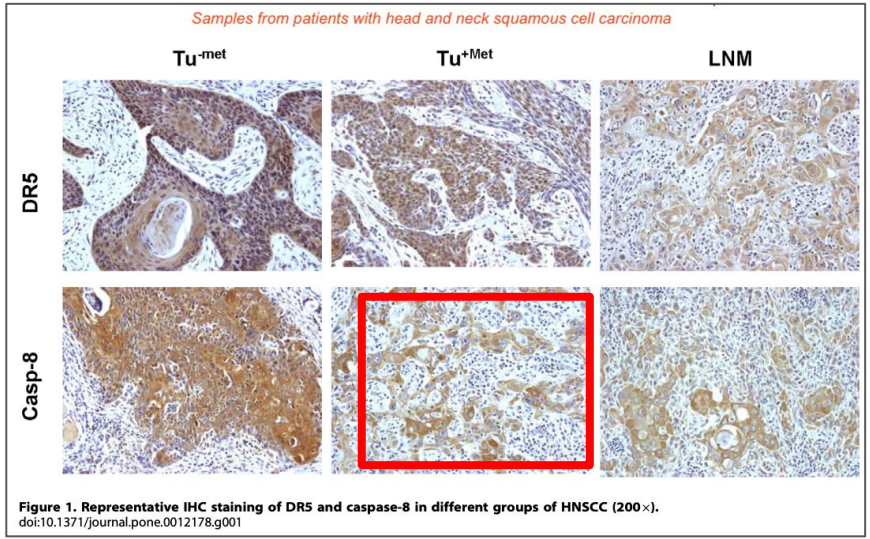


Figure 1. Representative IHC staining of DR5 and caspase-8 in different groups of HNSCC (200×).
doi:10.1371/journal.pone.0012178.g001

ARTICLE

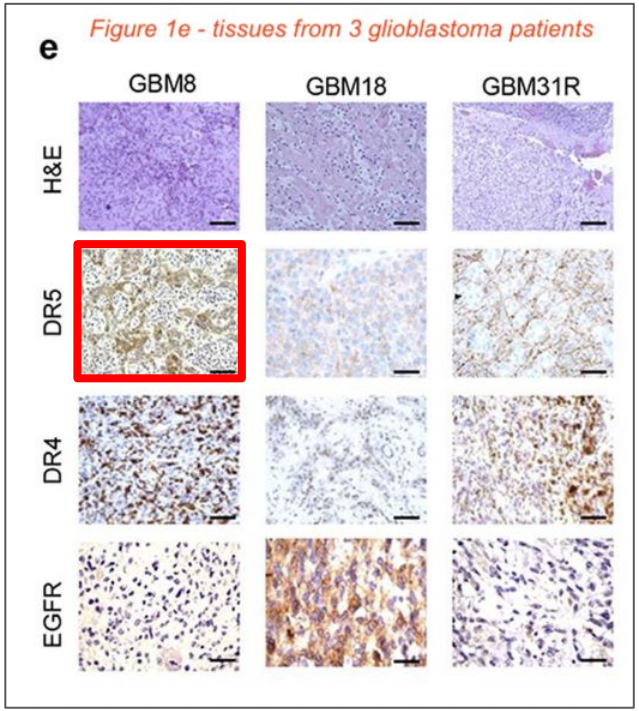
Nature Communications (2022)

https://doi.org/10.1038/s41467-022-30558-3 OPEN

Target receptor identification and subsequent treatment of resected brain tumors with encapsulated and engineered allogeneic stem cells

Deepak Bhere[1,2,13], Sung Hugh Choi[1,2], Pim van de Donk[1,3], David Hope[1,2], Kiki Gortzak[1,3], Amina Kunnummal[1,3], Jasneet Khalsa[1,2], Esther Revai Lechtich[1,2], Clemens Reinshagen[1,2], Victoria Leon[1,2], Nabil Nissar[1,3], Wenya Linda Bi[2], Cheng Feng[4], Hongbin Li[4], Yu Shrike Zhang[4], Steven H. Liang[3], Neil Vasdev[3], Walid Ibn Essayed[2], Pablo Valdes Quevedo[2], Alexandra Golby[2], Naima Banouni[5], Anna Palagina[6], Reza Abdi[5], Brian Fury[7], Stelios Smirnakis[6], Alarice Lowe[8,14], Brock Reeve[9], Arthur Hiller[10], E. Antonio Chiocca[2], Glenn Prestwich[11,15], Hiroaki Wakimoto[1,2,12], Gerhard Bauer[7] & Khalid Shah[1,2,9]✉

2022



Red boxes highlight a figure panel in the 2022 paper (right) that looks identical to a higher-resolution panel found in a 2010 paper (left) from a different group of researchers, where it represents tissue from a different patient, stained with a different antibody.

OPEN ACCESS Freely available online

PLOS ONE (2010)

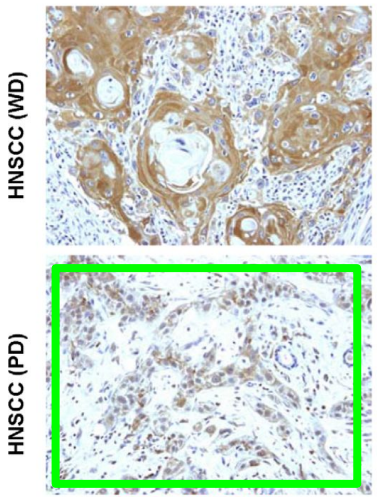
PLOS one

Analysis of Death Receptor 5 and Caspase-8 Expression in Primary and Metastatic Head and Neck Squamous Cell Carcinoma and Their Prognostic Impact

Heath A. Elrod[1], Songqing Fan[1], Susan Muller[2], Georgia Z. Chen[1], Lin Pan[3], Mourad Tighiouart[3], Dong M. Shin[1], Fadlo R. Khuri[1], Shi-Yong Sun[1]*

1 Department of Hematology and Medical Oncology, Emory University School of Medicine and Winship Cancer Institute, Atlanta, Georgia, United States of America, **2** Department of Pathology, Emory University School of Medicine and Winship Cancer Institute, Atlanta, Georgia, United States of America, **3** Department of Biostatistics and Bioinformatics, Emory University School of Medicine and Winship Cancer Institute, Atlanta, Georgia, United States of America

Green boxes highlight a figure panel in the 2022 paper (right) that looks identical to a higher-resolution panel found in a 2010 paper (left) from a different group of researchers, where it represents tissue from a different patient, stained with a different antibody.



Caspase-8 IHC staining
HNSCC: head and neck squamous cell carcinoma
WD: well-differentiated: PD, poorly-differentiated

Fig. 3. Comparison of DR5 (*A*) and caspase-8 (B) b differentiated (MD) HNSCC. Pictures are representative IH with paired *t* test.
doi:10.1371/journal.pone.0012178.g003

ARTICLE

Nature Communications (2022)

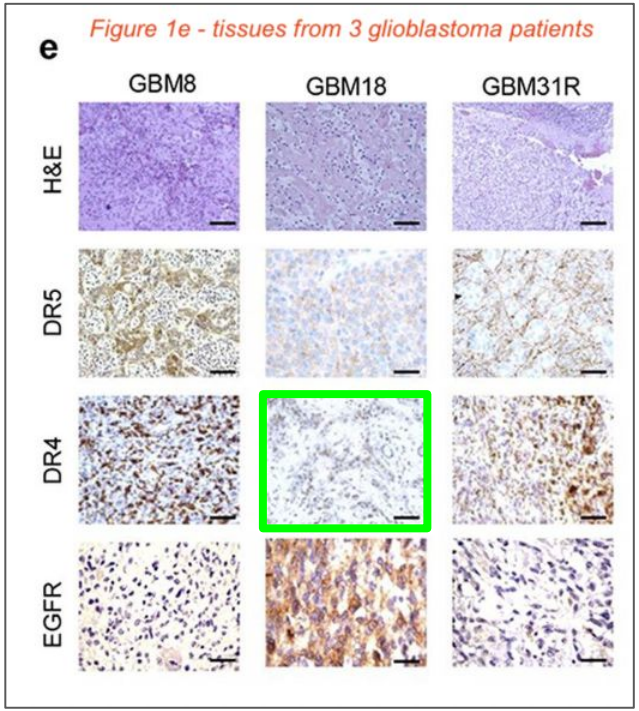
Check for updates

https://doi.org/10.1038/s41467-022-30558-3 OPEN

Target receptor identification and subsequent treatment of resected brain tumors with encapsulated and engineered allogeneic stem cells

Deepak Bhere[1,2,13], Sung Hugh Choi[1,2], Pim van de Donk[1,3], David Hope[1,2], Kiki Gortzak[1,3], Amina Kunnummal[1,3], Jasneet Khalsa[1,2], Esther Revai Lechtich[1,2], Clemens Reinshagen[1,2], Victoria Leon[1,2], Nabil Nissar[1,3], Wenya Linda Bi[2], Cheng Feng[4], Hongbin Li[4], Yu Shrike Zhang[4], Steven H. Liang[3], Neil Vasdev[3], Walid Ibn Essayed[2], Pablo Valdes Quevedo[2], Alexandra Golby[2], Naima Banouni[5], Anna Palagina[6], Reza Abdi[5], Brian Fury[7], Stelios Smirnakis[6], Alarice Lowe[8,14], Brock Reeve[9], Arthur Hiller[10], E. Antonio Chiocca[2], Glenn Prestwich[11,15], Hiroaki Wakimoto[1,2,12], Gerhard Bauer[7] & Khalid Shah[1,2,9]✉

2022

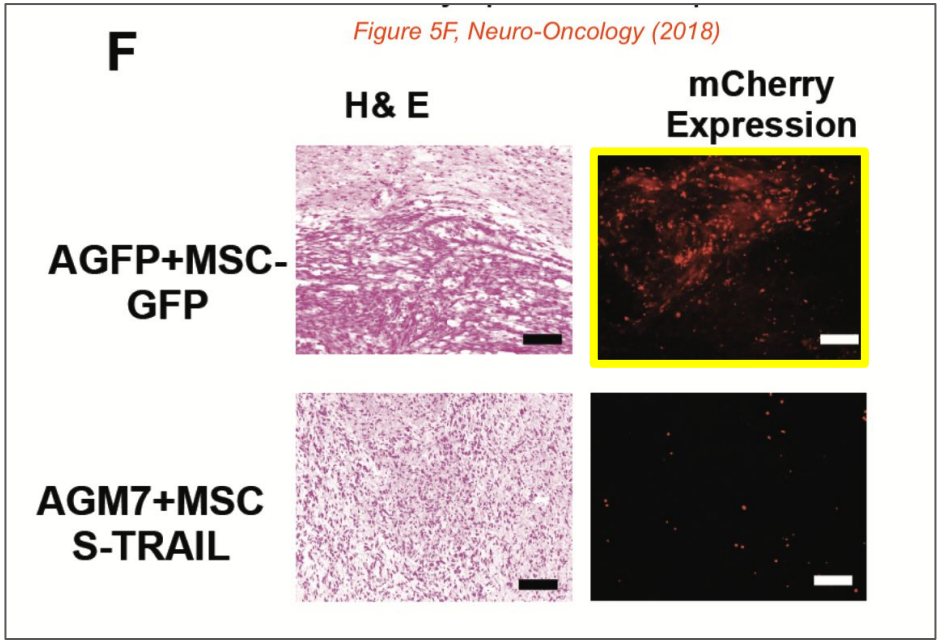


Neuro-Oncology
20(2), 215–224, 2018 | doi:10.1093/neuonc/nox138 | Advance Access date 25 July 2017

microRNA-7 upregulates death receptor 5 and primes resistant brain tumors to caspase-mediated apoptosis

Deepak Bhere,* Kaoru Tamura,* Hiroaki Wakimoto, Sung Hugh Choi, Benjamin Purow, Jeremy Debatisse, and Khalid Shah

Center for Stem Cell Therapeutics and Imaging (D.B., K.T., H.W., S.H.C., J.D., K.S.), Department of Radiology (D.B., K.T., H.W., S.H.C., J.D., K.S.), Department of Neurosurgery (H.W.), and Department of Neurology, Massachusetts General Hospital, Harvard Medical School, Boston, Massachusetts (K.S.); Department of Neurology, University of Virginia, Charlottesville, Virginia (B.P.); Harvard Stem Cell Institute, Harvard University, Cambridge, Massachusetts (K.S.); Center for Stem Cell Therapeutics and Imaging, Department of Neurosurgery, Brigham and Women's Hospital, Harvard Medical School, Boston, Massachusetts (D.B., H.W., S.H.C., K.S.)



ARTICLE
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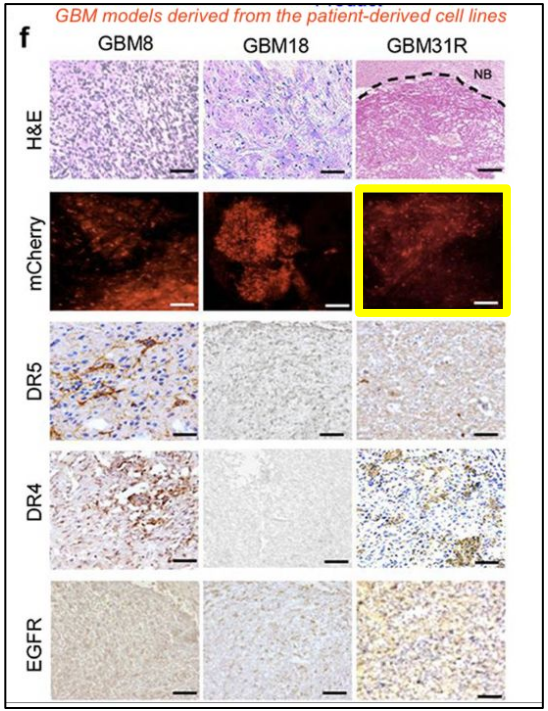
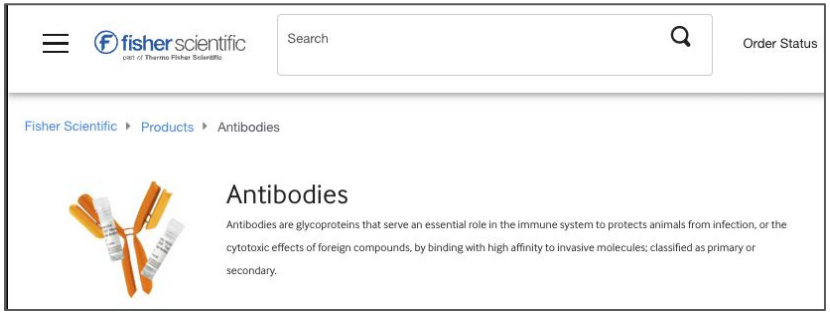
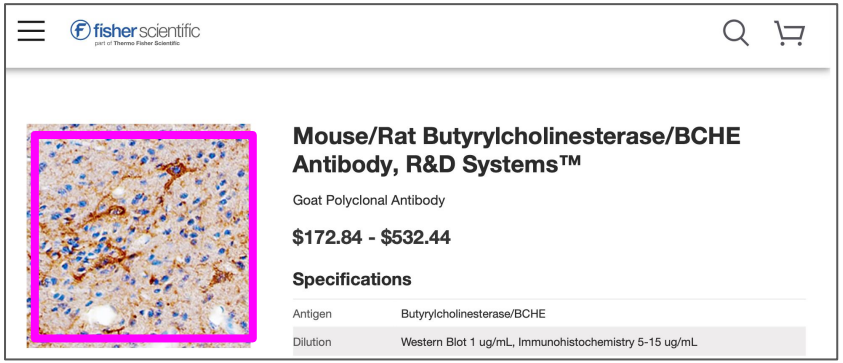


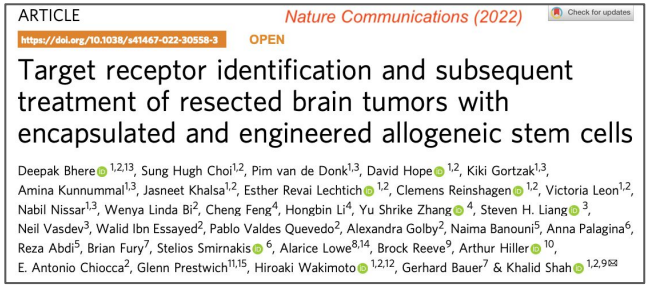
2022

Yellow boxes highlight a panel in Figure 1f in the 2022 paper (right) that looks identical to a higher-resolution panel found in a 2018 paper (left) from the same group of researchers. The different labels suggest that the panels represent different experiments.





Pink boxes highlight a panel in Figure 1f in the 2022 paper (right) that looks identical to a higher-resolution panel found on the website of Fisher Scientific, a vendor selling antibodies.
https://www.fishersci.com/shop/products/mouse-rat-butyrylcholinesterase-bche-antibody-r-d-systems/AF9024SP
The different labels (BCHE antibody vs. DR5) suggest that the panels represent different experiments.



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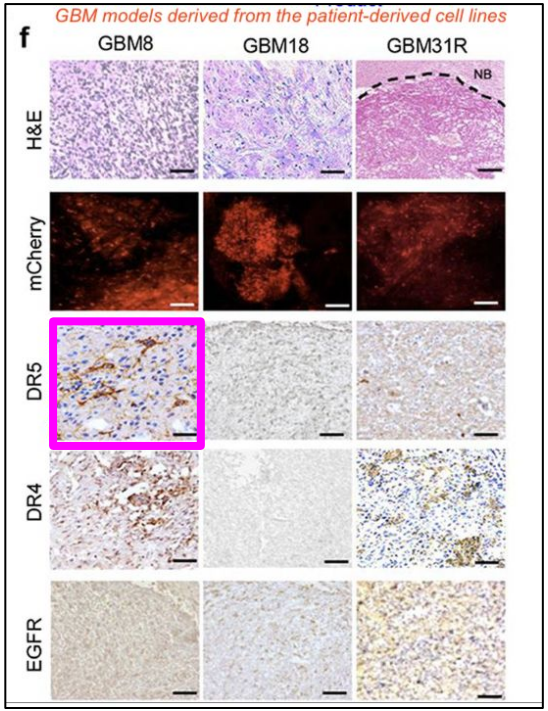

Kindy et al. J Transl Med (2016) 14:1
DOI 10.1186/s12967-015-0757-9

Journal of Translational Medicine

RESEARCH Open Access

A therapeutic cancer vaccine against GL261 murine glioma

Mark S. Kindy[1†], Jin Yu[1†], Hong Zhu[1], Michael T. Smith[2] and Sebastiano Gattoni-Celli[3,4*]

Immunoperoxidase staining of microglia in brain sections from mock-vaccinated and vaccinated mice.

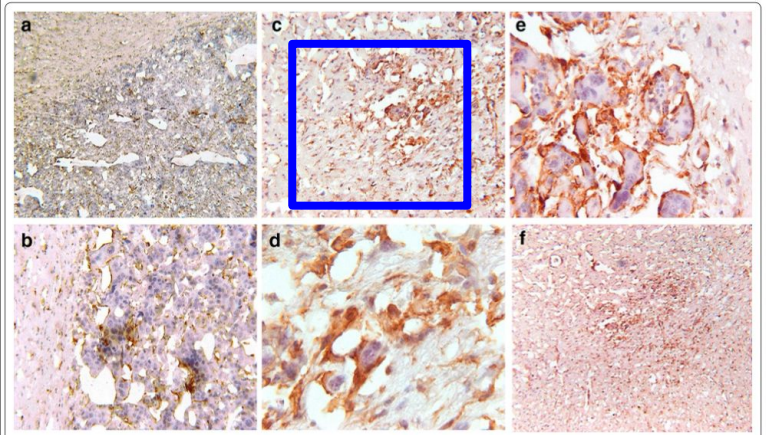

Fig. 5 Immunoperoxidase staining of microglia in brain sections from mock-vaccinated and vaccinated mice. **a** The large tumor from a mock-vaccinated mouse occupies the central, lower and right areas in this image. Uninvolved brain is in the *upper left quadrant* of the image. *Brown staining marks* microglia in the surrounding brain and in the tumor. The border of the tumor with the surrounding brain is distinct but ragged (magnification ×20). **b** This photomicrograph from the same mock-vaccinated mouse shows the interface of tumor and adjacent brain. Staining for microglia is present in both. Tumor cells are hyperchromatic and atypical mitoses are present. A thin rim of microglia staining adjacent to tumor cells is visible (magnification ×100). **c** This photomicrograph shows a much smaller tumor in a vaccinated mouse. Microglia cells are intermingled within the tumor, attached to tumor cells, and are more numerous in surrounding brain. Microglia cells are also more numerous around tumor cells in vaccinated mice compared to mock-vaccinated ones (magnification ×40). **d** In this photomicrograph from the same vaccinated mouse, numerous

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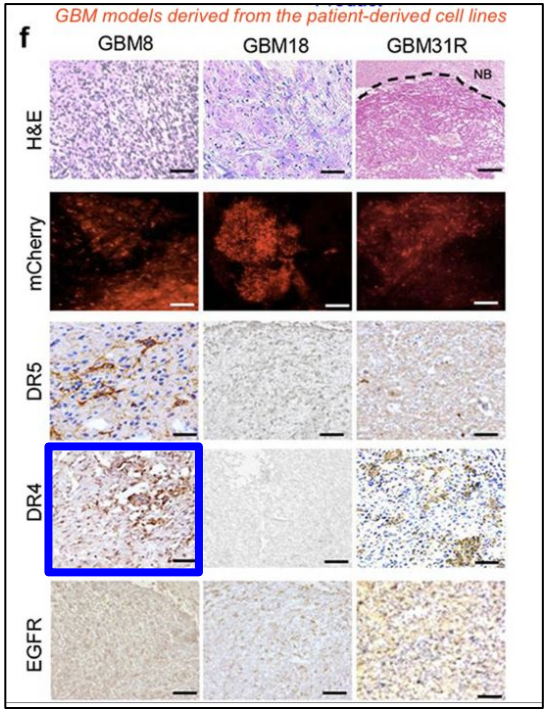
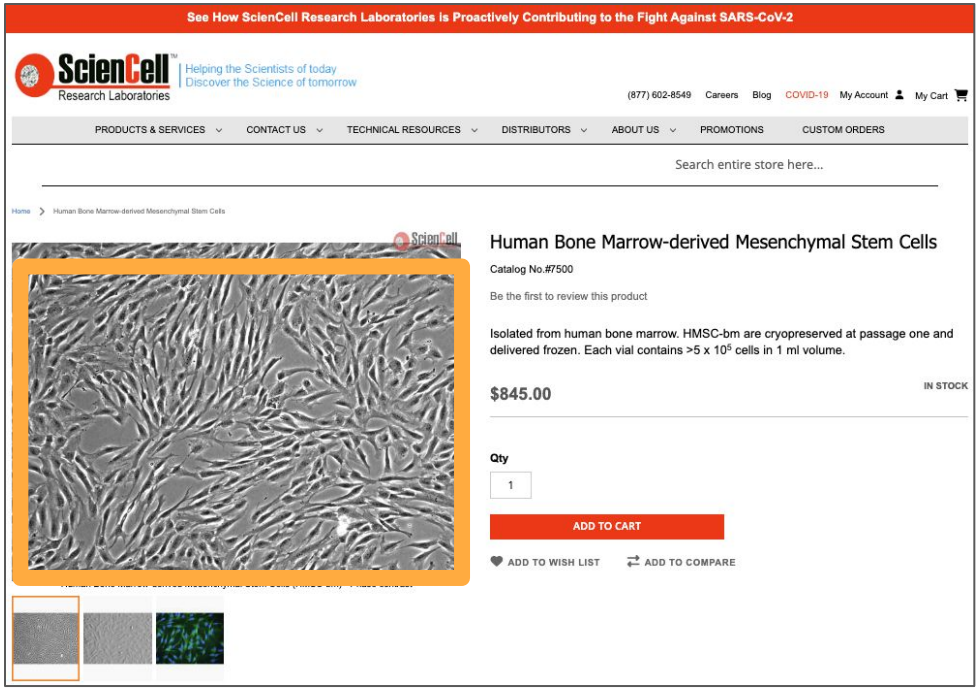
2022



Blue boxes highlight a panel in Figure 1f in the 2022 paper (right) that looks identical to a higher-resolution panel found in a 2016 paper (left) from the same group of researchers. The different labels suggest that the panels represent different experiments (e.g., mouse vs. patients).



Orange boxes highlight a panel in Supplemental Figure 2a in the 2022 paper (right) that looks identical to a higher-resolution panel found on the website of a vendor selling stem cells (left).
https://sciencellonline.com/human-bone-marrow-derived-mesenchymal-stem-cells/

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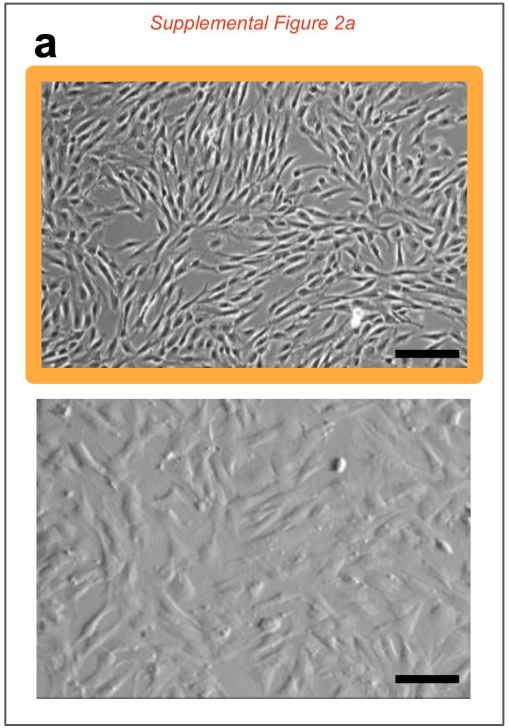

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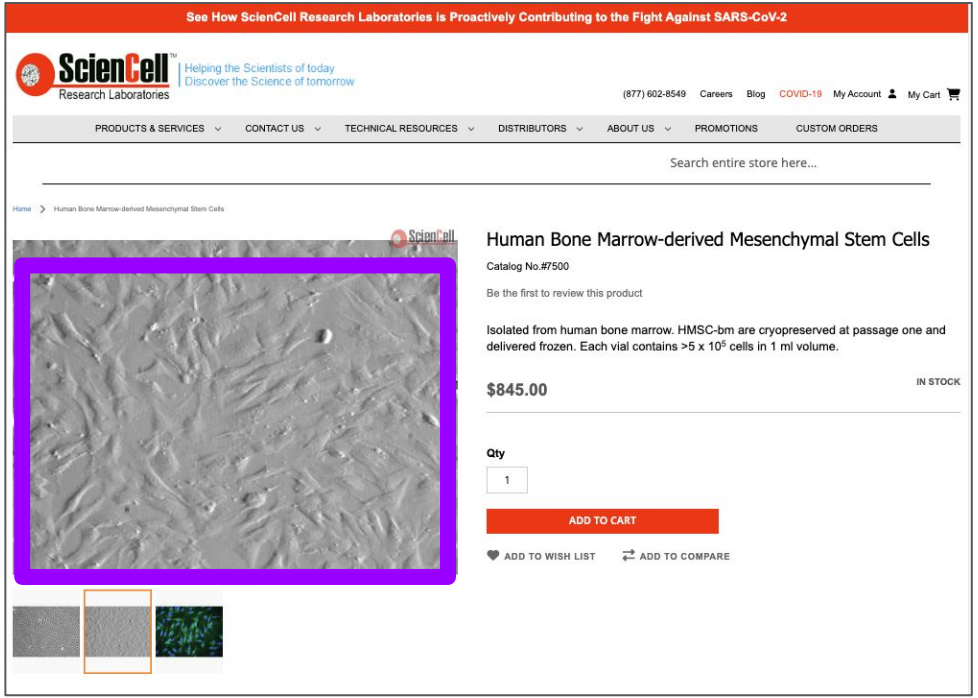

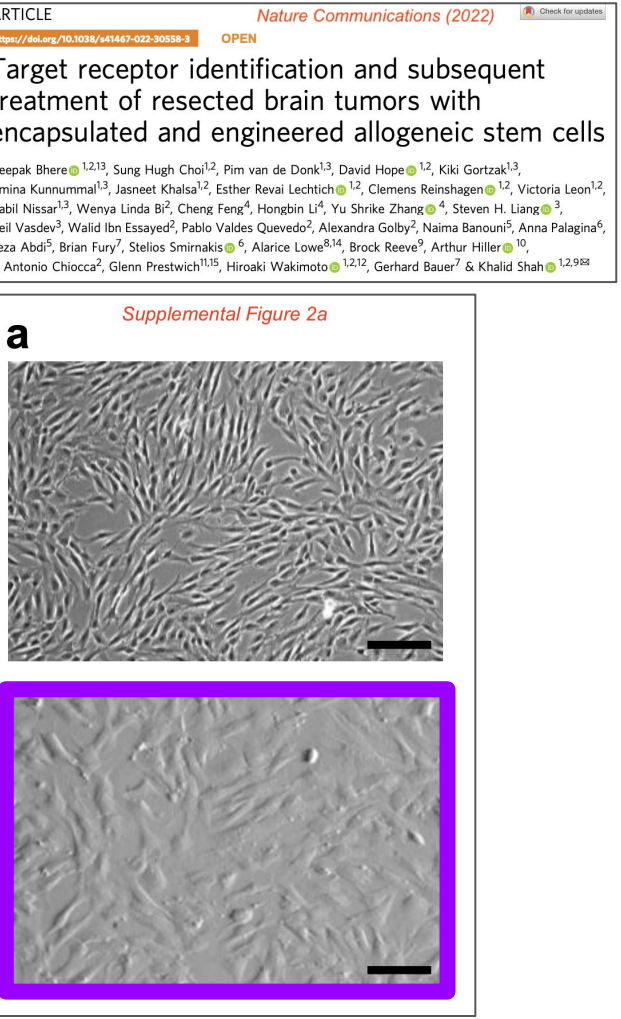

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Purple boxes highlight a panel in Supplemental Figure 2a in the 2022 paper (right) that looks identical to a higher-resolution panel found on the website of a vendor selling stem cells (left). https://sciencellonline.com/human-bone-marrow-derived-mesenchymal-stem-cells/

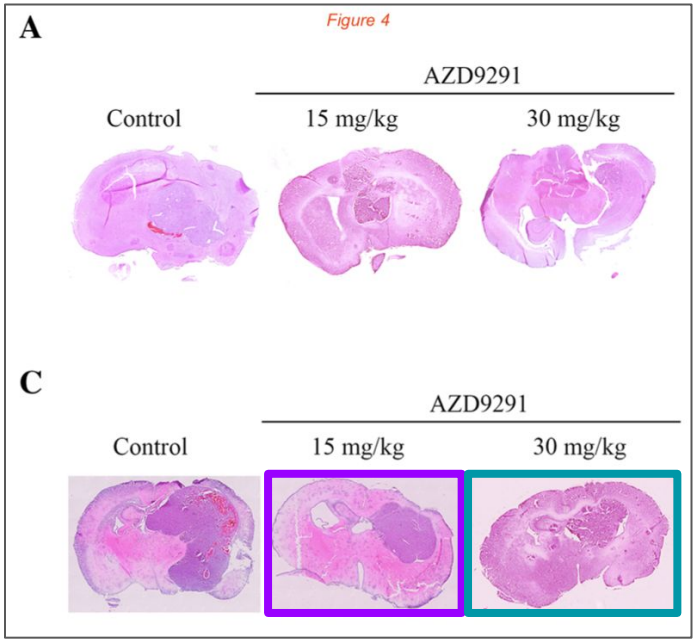
Liu et al. Journal of Experimental & Clinical Cancer Research (2019) 38:219
https://doi.org/10.1186/s13046-019-1235-7

Journal of Experimental & Clinical Cancer Research

RESEARCH Open Access

The third-generation EGFR inhibitor AZD9291 overcomes primary resistance by continuously blocking ERK signaling in glioblastoma

Xuejiao Liu[1,2†], Xiangyu Chen[1,2†], Lin Shi[1,2†], Qianqian Shan[1], Qiyu Cao[1], Chenglong Yue[4], Huan Li[1], Shengsheng Li[1], Jie Wang[1], Shangfeng Gao[1,2], Mingshan Niu[1,3*] and Rutong Yu[1,2*]



Purple and teal boxes highlight panels in Supplemental Figure 4b in the 2022 paper (right) that look identical to higher-resolution panels found in a 2019 paper (left) from a different group of researchers.
Both panels appear to be showing different experiments.

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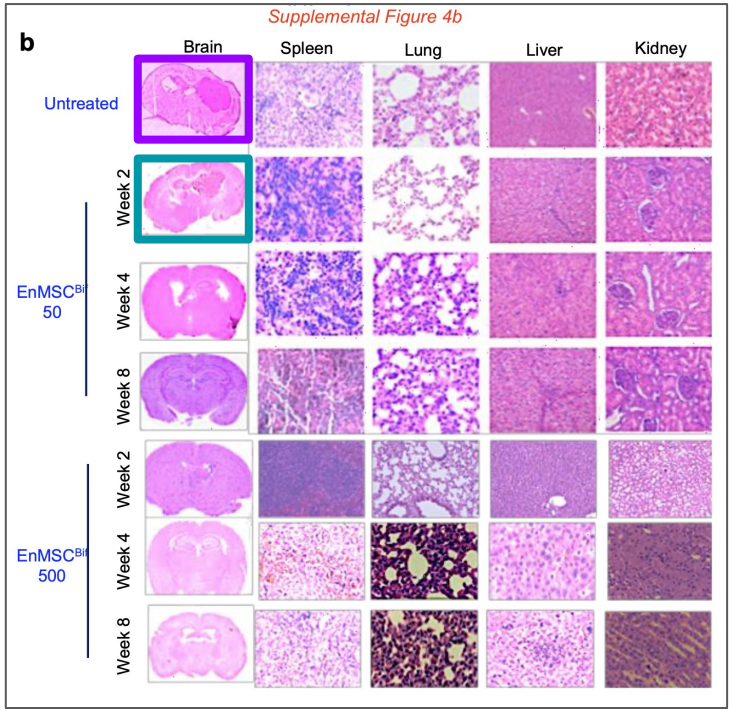

International Journal of Nanomedicine
Wang et al., DOI: 10.2147/IJN.S113882
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ORIGINAL RESEARCH

2016

The effect of dual-functional hyaluronic acid-vitamin E succinate micelles on targeting delivery of doxorubicin

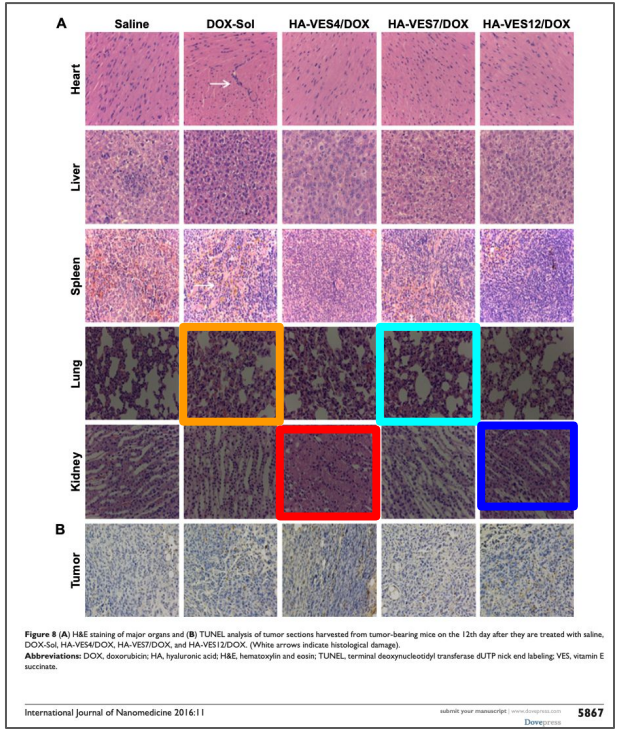

Figure 8 (**A**) H&E staining of major organs and (**B**) TUNEL analysis of tumor sections harvested from tumor-bearing mice on the 12th day after they are treated with saline, DOX-Sol, HA-VES4/DOX, HA-VES7/DOX, and HA-VES12/DOX. (White arrows indicate histological damage).
Abbreviations: DOX, doxorubicin; HA, hyaluronic acid; H&E, hematoxylin and eosin; TUNEL, terminal deoxynucleotidyl transferase dUTP nick end labeling; VES, vitamin E succinate.

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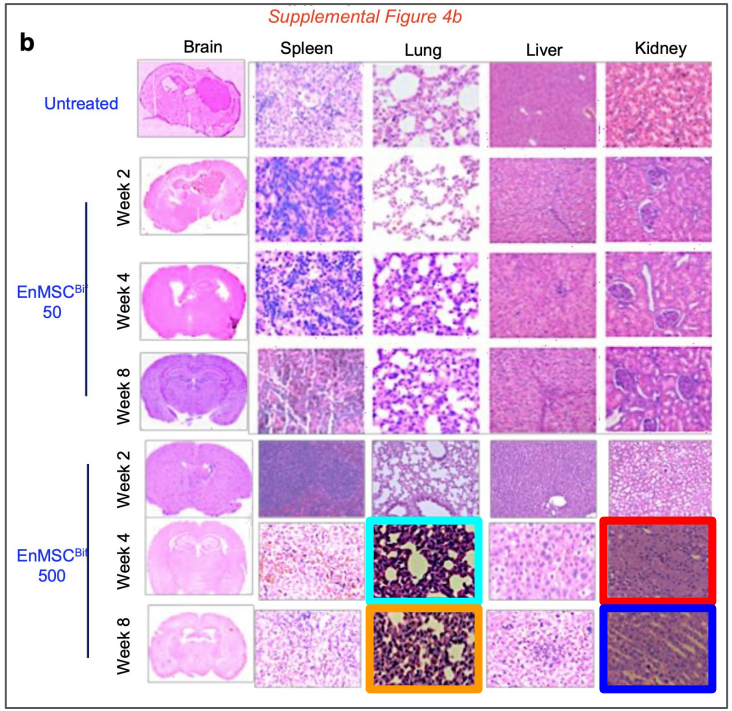
Cyan, orange, red, and blue boxes highlight panels in Supplemental Figure 4b in the 2022 paper (right) that look identical to higher-resolution panels found in a 2016 paper (left) from a different group of researchers.
The 2016 paper appears to be showing different experiments than the 2022 paper.



Hindawi Publishing Corporation
Neural Plasticity
Volume 2016, Article ID 3258494, 8 pages
http://dx.doi.org/10.1155/2016/3258494

Hindawi

Research Article

Synchrotron Radiation X-Ray Phase-Contrast Tomography Visualizes Microvasculature Changes in Mice Brains after Ischemic Injury

Peng Miao,[1] Zhixia Wu,[1] Miao Li,[1] Yuanyuan Ji,[1] Bohua Xie,[2] Xiaojie Lin,[2] and Guo-Yuan Yang[2]

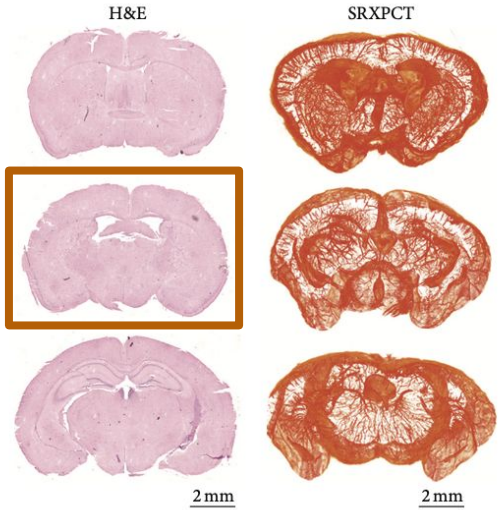


FIGURE 6: Comparison of H&E staining (a) and reconstructed sections of 3D SRXPCT (b) of mice brains.

Brown boxes highlight a panel in Supplemental Figure 4b in the 2022 paper (right) that looks identical to higher-resolution panels found in a 2016 paper (left) from a different group of researchers.

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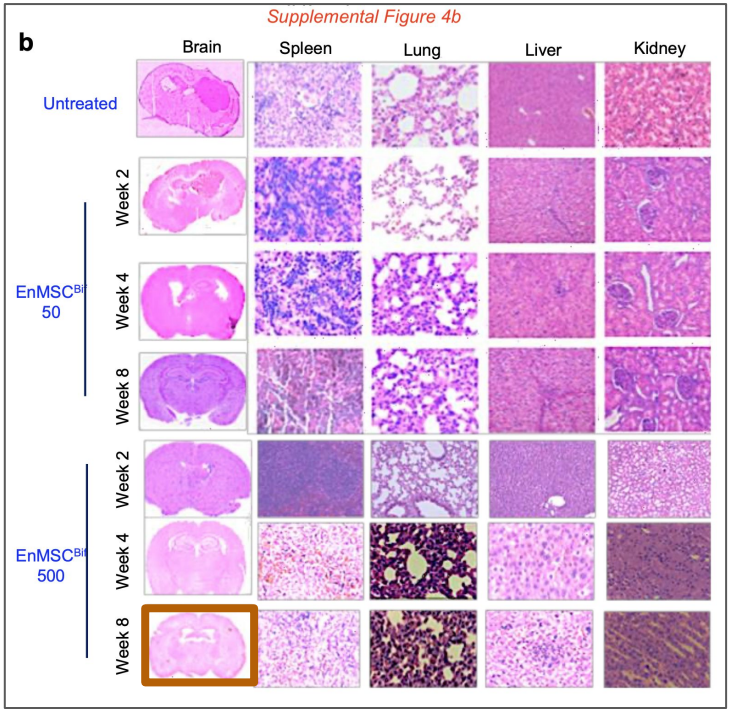

EXPERIMENTAL AND THERAPEUTIC MEDICINE 14: 5881-5888, 2017

Comparison of naturally aging and D-galactose induced aging model in beagle dogs

MUSI JI[1,2*], XIAOHUA SU[1*], JIZHEN LIU[1*], YI ZHAO[1], ZHIDONG LI[1], XUN XU[1], HUAWEN LI[1] and BAYAER NASHUN[1]

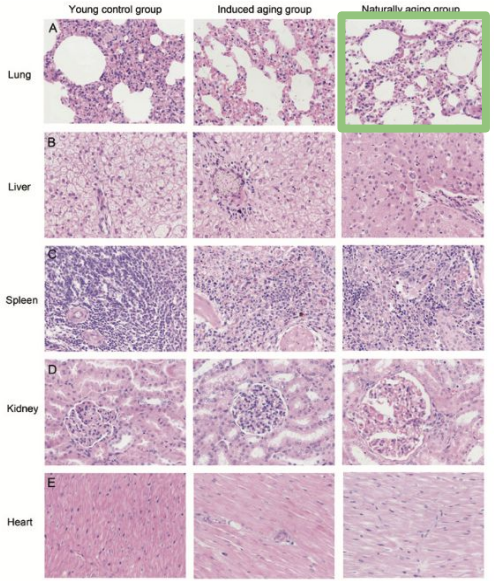

Figure 2. Histopathologic examinations on (A) lung, (B) liver, (C) spleen, (D) kidney and (E) heart at the end of the experiments (H&E staining, x40). H&E, hematoxylin and eosin.

Green boxes highlight a panel in Supplemental Figure 4b in the 2022 paper (right) that looks identical to a higher-resolution panel found in a 2017 paper (left) from a different group of researchers.

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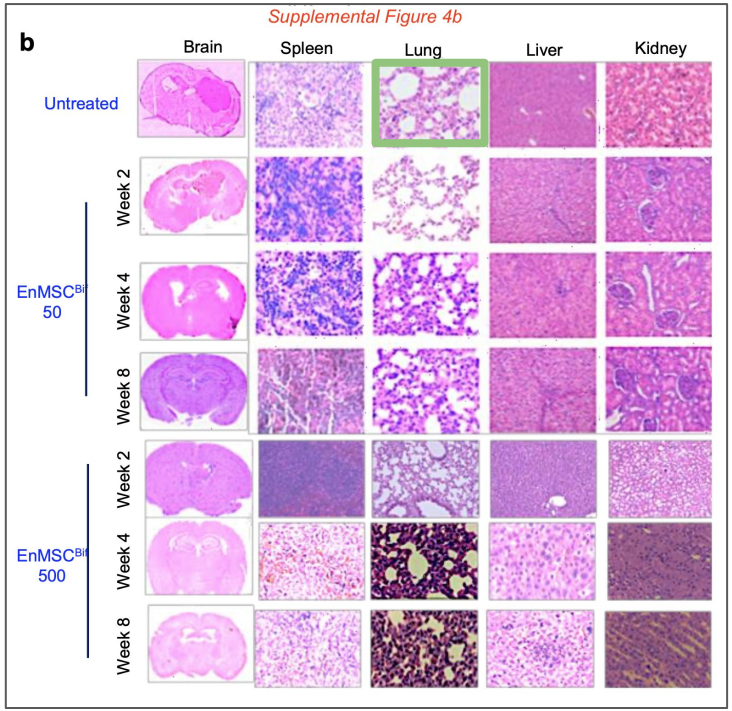
2022



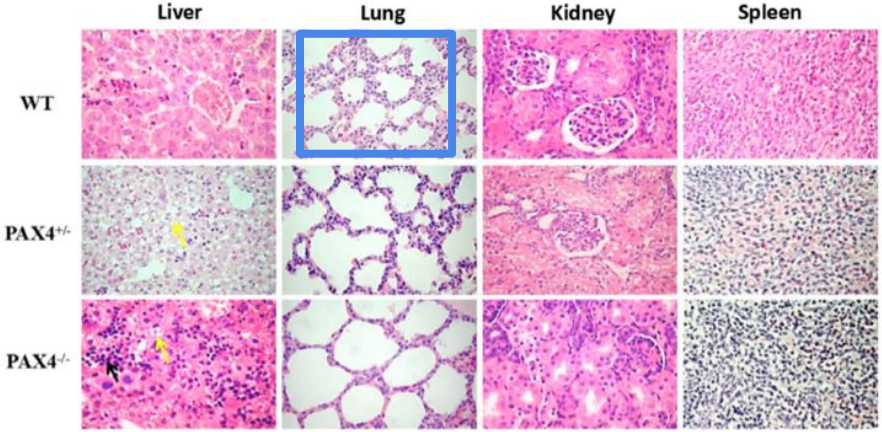
G3 Genes | Genomes | Genetics

Xu et al., G3 (2018), DOI: 10.1534/g3.118.300448

Generation and Phenotype Identification of *PAX4* Gene Knockout Rabbit by CRISPR/Cas9 System

Yuanyuan Xu,[1] Yong Wang,[1] Yuning Song, Jichao Deng, Mao Chen, Hongsheng Ouyang, Liangxue Lai,[2] and Zhanjun Li[2]
Jilin Provincial Key Laboratory of Animal Embryo Engineering, Jilin University, Changchun 130062, China

Figure 4, detail



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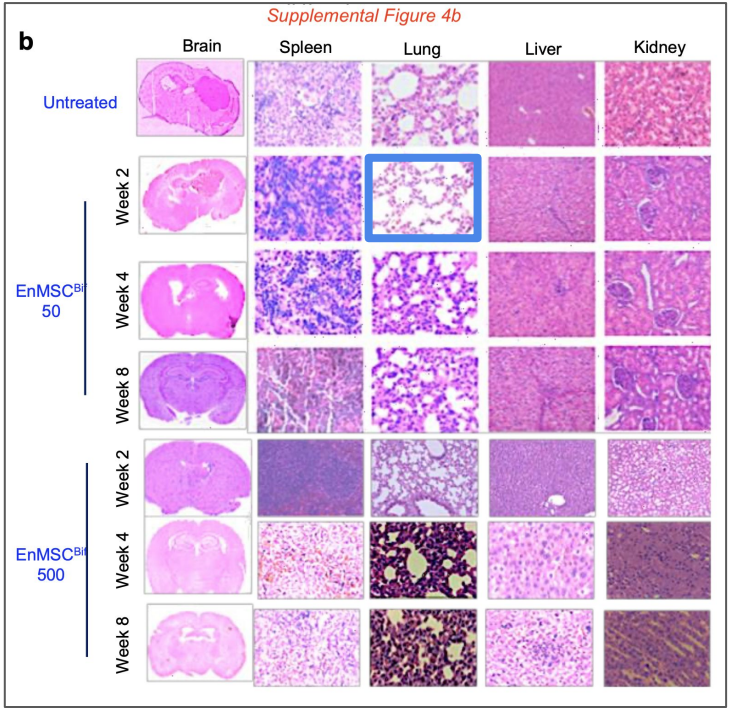
Target receptor identification and subsequent treatment of resected brain tumors with encapsulated and engineered allogeneic stem cells

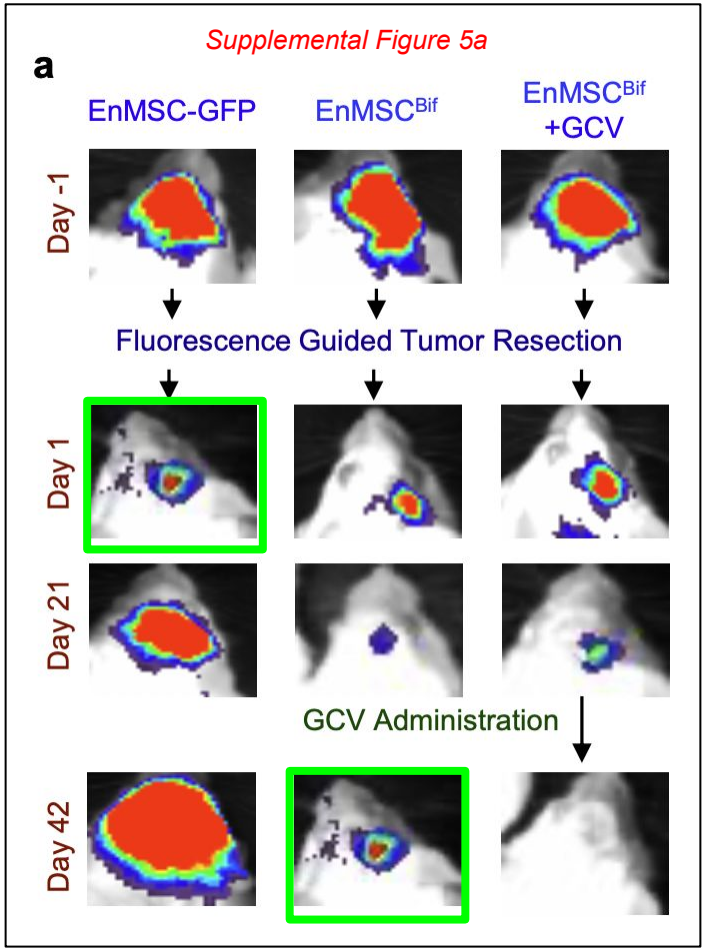
Deepak Bhere[1,2,13], Sung Hugh Choi[1,2], Pim van de Donk[1,3], David Hope[1,2], Kiki Gortzak[1,3], Amina Kunnummal[1,3], Jasneet Khalsa[1,2], Esther Revai Lechtich[1,2], Clemens Reinshagen[1,2], Victoria Leon[1,2], Nabil Nissar[1,3], Wenya Linda Bi[2], Cheng Feng[4], Hongbin Li[4], Yu Shrike Zhang[4], Steven H. Liang[3], Neil Vasdev[3], Walid Ibn Essayed[2], Pablo Valdes Quevedo[2], Alexandra Golby[2], Naima Banouni[5], Anna Palagina[6], Reza Abdi[5], Brian Fury[7], Stelios Smirnakis[6], Alarice Lowe[8,14], Brock Reeve[9], Arthur Hiller[10], E. Antonio Chiocca[2], Glenn Prestwich[11,15], Hiroaki Wakimoto[1,2,12], Gerhard Bauer[7] & Khalid Shah[1,2,9]✉

2022

Supplemental Figure 4b



Blue boxes highlight a panel in Supplemental Figure 4b in the 2022 paper (right) that looks identical to a higher-resolution panel found in a 2018 paper (left) from a different group of researchers.



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2022

Green boxes highlight two panels in Supplemental Figure 5a in the 2022 paper that look identical, even though they appear to show different time points and experimental treatments.

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RESEARCH ARTICLE

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Developing and characterizing a two-layered safety switch for cell therapies

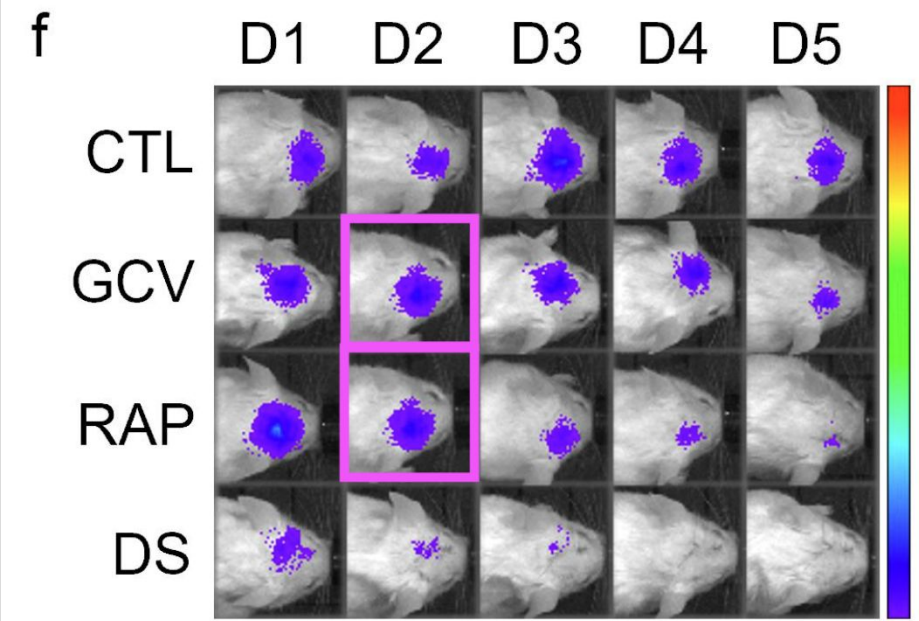
Filippo Rossignoli[a,b], Danielle Hoffman[a,b], Emaan Atif[a,b], and Khalid Shah[a,b,c]

[a]Center for Stem Cell and Translational Immunotherapy (CSTI), Harvard Medical School, Boston, MA, USA; [b]Department of Neurosurgery, Brigham and Women's Hospital, Boston, MA, USA; [c]Harvard Stem Cell Institute, Harvard University, Boston, MA, USA

2023

Pink boxes highlight two panels in Figure 3f that appear to show the same animal and luminescence pattern.

Figure 3f



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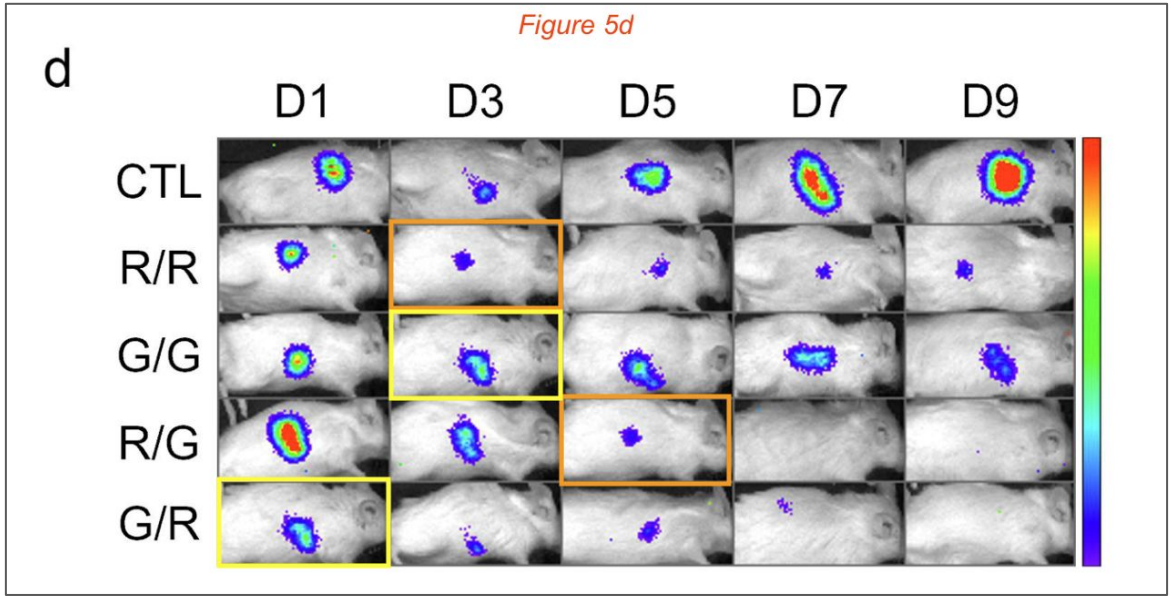
2023

Yellow and orange boxes highlight two sets of panels in Figure 5d that appear to show the same animal and luminescence pattern.



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Original Research

OXFORD

Fate and Efficacy of Engineered Allogeneic Stem Cells Targeting Cell Death and Proliferation Pathways in Primary and Brain Metastatic Lung Cancer

Susana Moleirinho[1,2], Yohei Kitamura[1,2], Paulo S.G.N. Borges[1,2], Sophia Auduong[1,2], Seyda Kilic[1,2], David Deng[1,2], Nobuhiko Kanaya[1,2], David Kozono[3], Jing Zhou[4], Jeffrey J. Gray[4], Esther Revai-Lechtich[1,2], Yanni Zhu[1], Khalid Shah*[1,2,5]

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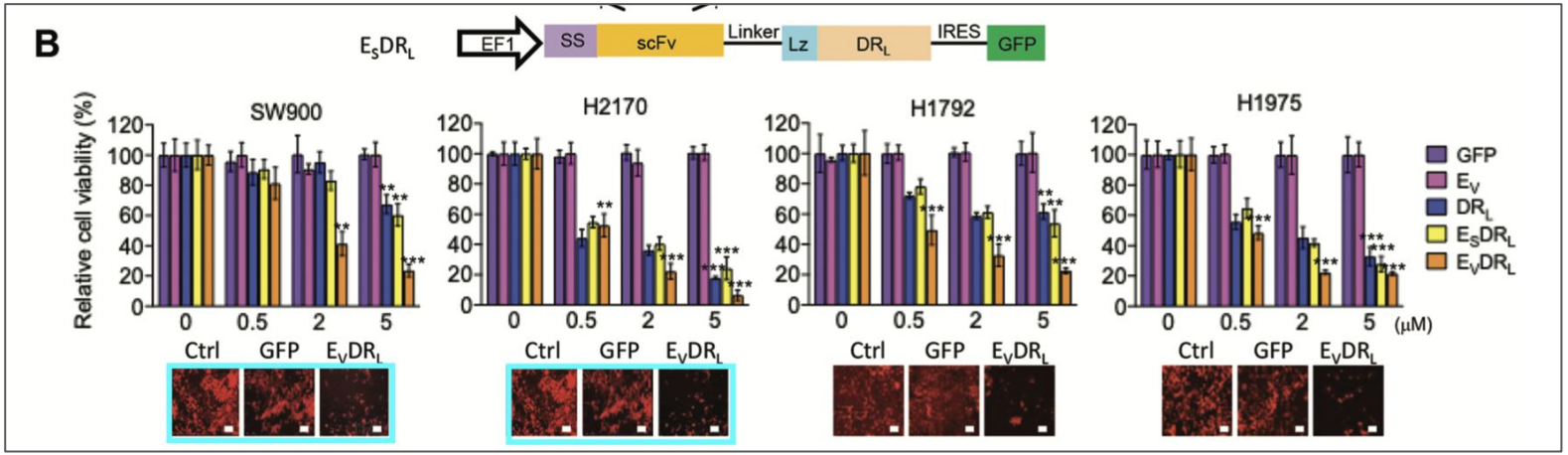
2023

Cyan boxes highlight two sets of panels in Figure 2B that appear to show the same photos.



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Establishment and immune phenotyping of patient-derived glioblastoma models in humanized mice

Longsha Liu[1,2], Thijs A. van Schaik[1,2], Kok-Siong Chen[1,2], Filippo Rossignoli[1,2], Paulo Borges[1,2], Vladimir Vrbanac[3], Hiroaki Wakimoto[1,2,4] and Khalid Shah[1,2,5]*

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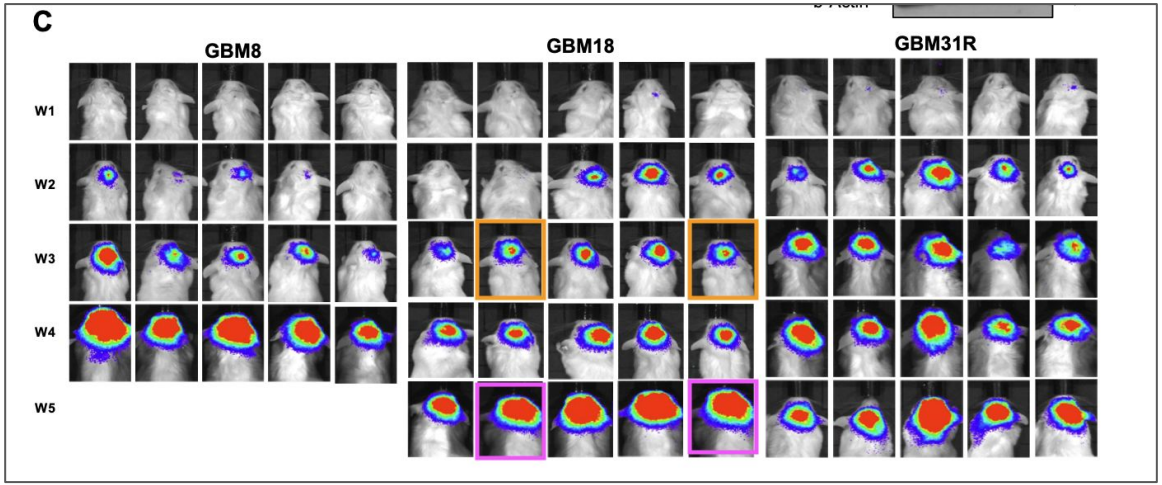

2023

Supplemental Figure 1C:
Orange boxes: Two panel in the GBM18/Week 3 row appear to show the same mouse
Pink boxes: Two panel in the GBM18/Week 5 row appear to show the same mouse