B16F10.9 Cell Line

(No. M7-2179)

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Overview

Spontaneous metastatic spread of melanoma after surgery of the primary tumor. immunization against melanoma metastasis

Relevance: A model to study melanoma metastasis in mice.

Melanoma metastatic spread, immunization against melanoma metastasis

Relevant disease: melanoma

Parental Line: B16F I0

Host: Mouse

Tissue: skin

Relevance: A model to study melanoma metastasis in mice.

References

Porgador A, Brenner B, Vadai E, Feldman M, Eisenbach L. Immunization by gamma-IFN-treated B16-F10.9 melanoma cells protects against metastatic spread of the parental tumor. Int J Cancer. 1991;47(S6):54-60. doi:10.1002/ijc.2910470713 Â

Overexpression of a set of genes, including WISP-1, common to pulmonary metastases of both mouse D122 Lewis lung carcinoma and B16-F10.9 melanoma cell lines.

Margalit O, Eisenbach L, Amariglio N, Kaminski N, Harmelin A, Pfeffer R, Shohat M, Rechavi G, Berger R. Br J Cancer. 2003 Jul 21;89(2):314-9. doi: 10.1038/sj.bjc.6600977. PMID: 12865923 Free PMC article.

To define genetic determinants of pulmonary metastases, we have applied oligonucleotide microarrays to established murine models of highly metastatic D122 Lewis lung carcinoma and B16-F10.9 melanoma cell lines. These models are characterised by primary subcutaneous â€

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Unmasking by soluble IL-6 receptor of IL-6 effect on metastatic melanoma: growth inhibition and differentiation of B16-F10.9 tumor cells.

Oh JW, Katz A, Harroch S, Eisenbach L, Revel M, Chebath J. Oncogene. 1997 Jul 31;15(5):569-77. doi: 10.1038/sj.onc.1201213. PMID: 9247310

The in vitro IL-6 response can be restored in the highly metastatic melanoma B16-F10.9 by addition of recombinant soluble IL-6 receptor alpha-chain (sIL-6R). The F10.9 cells then undergo irreversible growth-arrest and show increased adherence with chan â€

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Combined vaccination with major histocompatibility class I and interleukin 2 gene-transduced melanoma cells synergizes the cure of postsurgical established lung metastases.

Porgador A, Tzehoval E, Vadai E, Feldman M, Eisenbach L. Cancer Res. 1995 Nov 1;55(21):4941-9. PMID: 7585534

Mice given injections intrafootpad of tumorigenic doses of transduced clones manifested significantly reduced postsurgical spontaneous metastasis. After i.v. inoculation, mice given injections of F10.9-Kb expressors did not develop experimental lung metastases; mice â€

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Effective anti-metastatic melanoma vaccination with tumor cells transfected with MHC genes and/or infected with Newcastle disease virus (NDV).

Plaksin D, Porgador A, Vadai E, Feldman M, Schirrmacher V, Eisenbach L. Int J Cancer. 1994 Dec 15;59(6):796-801. doi: 10.1002/ijc.2910590615. PMID: 7989121

The therapeutic efficacy of active immunization with B16-F10.9 melanoma cells transfected with syngeneic major histocompatibility complex (MHC) class-I genes, modified by infection with Newcastle Disease virus (NDV) or modified by both treatments, was compared. B16- â€

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c-fos and c-jun overexpression in malignant cells reduces their tumorigenic and metastatic potential, and affects their MHC class I gene expression.

Yamit-Hezi A, Plaksin D, Eisenbach L. Oncogene. 1994 Apr;9(4):1065-79. PMID: 8134110

Transfection of c-jun and c-fos genes into the high metastatic clones D122 (3LL) and F10.9 (B16 melanoma) resulted in activation of H-2 class I gene expression. D122 transfectants expressing high levels of c-jun and c-fos and F10.9 transfectants expres â€

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Amplification of the expression of major histocompatibility class-I antigens by inducers of differentiation and gamma interferon in murine and human solid tumor-cell lines.

Fenig E, Nordenberg J, Lurie H, Angel P, Feldman M, Eisenbach L. Int J Oncol. 1993 Feb;2(2):279-82. doi: 10.3892/ijo.2.2.279. PMID: 21573551

Treatment of B16 F10-9 mouse melanoma cell line and RC-29 human renal carcinoma cell line with chemical inducers of differentiation such as sodium butyrate (SB) hexamethylene bisacetamide (HMBA) and L-histidinol significantly increased the expression of major histoc â€l

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Abrogation of B16 melanoma metastases by long-term low-dose interleukin-6 therapy.

Katz A, Shulman LM, Porgador A, Revel M, Feldman M, Eisenbach L. J Immunother Emphasis Tumor Immunol. 1993 Feb;13(2):98-109. doi: 10.1097/00002371-199302000-00004. PMID: 8318501

IL-6 therapy could be started even 10 days after tumor injection, when metastases are already established. Moreover, IL-6 treatment of mice bearing F10.9 tumors in the footpads resulted in complete protection against pulmonary spontaneous metastasis and in long-term â€

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Immunization by gamma-IFN-treated B16-F10.9 melanoma cells protects against metastatic spread of the parental tumor.

Porgador A, Brenner B, Vadai E, Feldman M, Eisenbach L. Int J Cancer Suppl. 1991;6:54-60. doi: 10.1002/ijc.2910470713. PMID: 1906054

B16-F10.9 is a highly metastatic clone of the B16-F10 melanoma line, that expresses low levels of MHC class-I antigens. ...Immunization with both the positive transfectant KI and the gamma-IFN-treated F10.9 cells protected in vivo against metast â€

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H-2Kb transfection of B16 melanoma cells results in reduced tumourigenicity and metastatic competence.

Porgador A, Feldman M, Eisenbach L. J Immunogenet. 1989 Aug-Oct;16(4-5):291-303. doi: 10.1111/j.1744-313x.1989.tb00475.x. PMID: 2639904

The metastatic B16 mouse melanoma shows a low cell surface expression of H-2Kb and H-2Db class I antigens on cells of both the high-metastatic line B16-F10 and the low-metastatic line B16-F1. Similarly, newly generated clones of these lines, having different metastatic pro â€

Top of Form

Bottom of Form

B16-F10.9 or B16F10.9

mRNA-transfected Dendritic Cells Expressing Polypeptides That Link MHC-I Presentation to Constitutive TLR4 Activation Confer Tumor Immunity.

Cafri G, Sharbi-Yunger A, Tzehoval E, Alteber Z, Gross T, Vadai E, Margalit A, Gross G, Eisenbach L. Mol Ther. 2015 Aug;23(8):1391-1400. doi: 10.1038/mt.2015.90. Epub 2015 May 22. PMID: 25997427 Free PMC article.

This superiority was also evident in the ability to protect mice from tumor progression following the administration of B16F10.9 melanoma cells and to inhibit the development of pre-established B16F10.9 tumors. ...

2

Recognition and prevention of tumor metastasis by the NK receptor NKp46/NCR1.

Glasner A, Ghadially H, Gur C, Stanietsky N, Tsukerman P, Enk J, Mandelboim O. J Immunol. 2012 Mar 15;188(6):2509-15. doi: 10.4049/jimmunol.1102461. Epub 2012 Feb 3. PMID: 22308311 Free article.

To address this question, we studied the activity of the NK cell receptor NKp46/NCR1 in two spontaneous metastasis models, the B16F10.9 melanoma (B16) and the Lewis lung carcinoma (D122) in the NCR1 knockout mouse that was generated by our group, in various i â€

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Improving postoperative immune status and resistance to cancer metastasis: a combined perioperative approach of immunostimulation and prevention of excessive surgical stress responses.

Goldfarb Y, Sorski L, Benish M, Levi B, Melamed R, Ben-Eliyahu S. Ann Surg. 2011 Apr;253(4):798-810. doi:

10.1097/SLA.0b013e318211d7b5. PMID: 21475023

Blood was withdrawn, marginating-pulmonary leukocytes were harvested, and NK activity and lung MADB106 tumor retention were assessed. In addition, C57BL/6 mice were implanted with syngeneic B16F10.9 melanoma cells. When tumors reached 100 mm, mice were treated with â€

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Improving survival rates in two models of spontaneous postoperative metastasis in mice by combined administration of a beta-adrenergic antagonist and a cyclooxygenase-2 inhibitor.

Glasner A, Avraham R, Rosenne E, Benish M, Zmora O, Shemer S, Meiboom H, Ben-Eliyahu S. J Immunol. 2010 Mar 1;184(5):2449-57. doi: 10.4049/jimmunol.0903301. Epub 2010 Feb 1. PMID: 20124103 Free article.

In this study, in mice undergoing primary tumor excision, we tested the survival-enhancing potential of perioperative blockade of catecholamines and prostaglandins, and studied potential mediating mechanisms. C57BL/6J mice were inoculated intrafootpad with syngeneic B16F10 â€

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Tumor-targeted hyaluronan nanoliposomes increase the antitumor activity of liposomal Doxorubicin in syngeneic and human xenograft mouse tumor models.

Peer D, Margalit R. Neoplasia. 2004 Jul-Aug;6(4):343-53. doi: 10.1593/neo.03460. PMID: 15256056 Free PMC article.

The tHA-LIP were long-circulating, more than all controls, in healthy and tumor-bearing (C57BL/6/B16F10.9; BALB/c/C-26) mice. Mediated by tHA-LIP, DXR accumulation in tumor-bearing lungs was 30-, 6.7-, and 3.5-fold higher than free DXR, nt-LIP, and Doxil, respective â€

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Loading mitomycin C inside long circulating hyaluronan targeted nano-liposomes increases its antitumor activity in three mice tumor models.

Peer D, Margalit R. Int J Cancer. 2004 Feb 20;108(5):780-9. doi: 10.1002/ijc.11615. PMID: 14696107 Free article.

In 3 tumor models, BALB/c bearing C-26 solid tumors; C57BL/6 bearing B16F10.9 or (separately) D122 lung metastasis, tHA-LIP were long-circulating, 7-fold and 70-fold longer than nt-LIP and free MMC, respectively. tHA-LIP-mediated MMC accumulation in tumor-bearing lu â€l

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Antiangiogenic gene therapy targeting the endothelium-specific receptor tyrosine kinase Tie2.

Lin P, Buxton JA, Acheson A, Radziejewski C, Maisonpierre PC, Yancopoulos GD, Channon KM, Hale LP, Dewhirst MW, George SE, Peters KG. Proc Natl Acad Sci U S A. 1998 Jul 21;95(15):8829-34. doi: 10.1073/pnas.95.15.8829. PMID: 9671764 Free PMC article.

Administration of AdExTek to mice with two different well established primary tumors, a murine mammary carcinoma (4T1) or a murine melanoma (B16F10.9), significantly inhibited the growth rate of both tumors (64% and 47%, respectively). ...

9

Novel anticancer prodrugs of butyric acid. 2.

Nudelman A, Ruse M, Aviram A, Rabizadeh E, Shaklai M, Zimrah Y, Rephaeli A. J Med Chem. 1992 Feb 21;35(4):687-94. doi: 10.1021/jm00082a009. PMID: 1542095

