

CLINICAL TRIAL

The Impact of M1/M2 Tumor Associated Macrophage (TAM) Polarization on Cancer Progression and Prognosis Prediction

First received on June 1, 2008. Last updated on June 3, 2008.

Purpose

The purpose of this study is to evaluate the correlation between M1/M2 phenotype of tumor associated macrophage (TAM) in lung cancer patients and clinical outcome.

Status	Recruiting
Condition	Tumor
Phase	N/A
Study Type	Observational
Study Design	Observational Model: Case Control, Time Perspective: Prospective
Official Title	The Impact of M1/M2 Tumor Associated Macrophage (TAM) Polarization on Cancer Progression and Prognosis Prediction

Further study details (as provided by National Institutes of Health Clinical Center (CC))

Enrollment	100
Start Date	September 2007

Detailed Description

Inflammatory response in the tumor micro-environment may facilitate the metastatic process (1). Macrophages are pivotal members of the inflammatory cells and the innate immune system within the tumor stroma. Tumor-associated macrophages can release growth factors, cytokines and inflammatory mediators that may facilitate cancer cell invasion, migration, angiogenesis, tumor progression or metastasis (1-5). A lot of studies showed TAM encounter factors that most frequently polarize them toward M2 type macrophage (1,4-5). It is interesting that in vitro studies macrophages have the potential to kill tumor by appropriate stimulation but these macrophage belonged to M1 and were not present in most tumor tissue (6). Some drugs target to suppress TAM have the promising results in animal models

(7-9). Switching the TAM phenotype from M2 to M1 may promote anti-tumor activity (10). In this study we will correlate TAM M1/M2 ratio and patients' prognosis, the gene expression pattern of TAM.

References 1. Coussens LM, Werb Z. Inflammation and cancer. *Nature* 2002;420(6917):860-867. 2. Crowther M, Brown NJ, Bishop ET, Lewis CE. Microenvironmental influence on macrophage regulation of angiogenesis in wounds and malignant tumors. *J Leukoc Biol* 2001;70(4):478-490. 3. Lin EY, Nguyen AV, Russell RG, Pollard JW. Colony-stimulating Factor 1 Promotes Progression of Mammary Tumors to Malignancy. *J. Exp. Med.* 2001;193(6):727-740. 4. Mantovani A. Cancer Inflammation by remote control. *Nature* 2005;435(7043):752-753. 5. Pollard JW. Tumor-educated macrophages promote tumour progression and metastasis. *Nature Reviews Cancer* 2004;4(1):71-78. 6. Sica A, Schippa T, Mantovani A, Allavena P. Tumor-associated macrophage are distinct M2 polarized population promoting tumor progression: potential targets of anti-tumor therapy. *Eur J of Cancer* 2006;42:717-27 7. Sessa C, De Braud F, Perotti A, et al. Trabectedin for women with ovarian carcinoma after treatment with platinum and taxanes fails. *J Clin Oncol* 2005;23:1867-74. 8. Wahl L, Kleinman HK. Tumor-associated macrophages as targets for cancer therapy. *J Natl Cancer Inst* 1998;90:1583-4. 9. Giraudo E, Inoue M, Hanahan D. An amino-bisphosphonate targets MMP-9-expressing macrophages and angiogenesis to impair cervical carcinogenesis. *J Clin Invest* 2004;114:623-33. 10. Guiducci C, Vicari AP, Sangaletti S, Trinchieri G, Colombo MP. Redirecting in vivo elicited tumor infiltrating macrophages and dendritic cells towards tumor rejection. *Cancer Res* 2005;65:3437-46.

Eligibility

Minimum Age Eligible for Study:	18 Years
Maximum Age Eligible for Study:	90 Years
Genders Eligible for Study:	Both

Criteria

Inclusion Criteria: - lung cancer with malignant pleural effusions Exclusion Criteria: - None

Contacts and Locations

Please refer to this study by its ClinicalTrials.gov identifier: NCT00690261

Locations

National Taiwan University Hospital

Status:	Recruiting
Facility:	Taipei, 100, Taiwan

Sponsors and Collaborators

National Taiwan University Hospital

More Information

Other Publications

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Health Authority:	Taiwan: Department of Health

Information obtained from ClinicalTrials.gov on February 08, 2012 [Link to the current ClinicalTrials.gov record.](http://clinicaltrials.gov/show/NCT00690261)
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