50~54.封闭B7-H1分子对肿瘤浸润树突状细胞介导T细胞免疫功能的影响[J].陈雅敏,梁晓华,黄若凡,周鑫莉.中国肿瘤生物治疗杂志,2009,16(1)

封闭B7-H1分子对肿瘤浸润树突状细胞介导T细胞免疫功能的影响

点此下载全文

陈雅敏 梁晓华 黄若凡 周鑫莉

复旦大学 附属华山医院 肿瘤科; 复旦大学 上海医学院 肿瘤学系和内科学系，上海 200040;复旦大学 附属华山医院 肿瘤科; 复旦大学 上海医学院 肿瘤学系和内科学系，上海 200040;复旦大学 附属华山医院 肿瘤科; 复旦大学 上海医学院 肿瘤学系和内科学系，上海 200040;复旦大学 附属华山医院 肿瘤科; 复旦大学 上海医学院 肿瘤学系和内科学系，上海 200040

基金项目：复旦大学附属华山医院科研基金\[No.145\]

DOI：10.3872/j.issn.1007-385X.2009.1.011

摘要：

目的:研究肿瘤浸润树突状细胞（tumor infiltrating dendritic cell，TIDC）及脾脏树突状细胞（splenic dendritic cell，SDC）表面B7 H1、B7 1、B7 2分子的表达情况；探讨封闭TIDC及SDC表面B7 H1分子对其介导T细胞免疫功能的影响。方法： CD11c磁珠阳性分选法提取荷瘤小鼠的TIDC及SDC，流式细胞术检测其表面B7 H1、B7 1、B7 2分子的表达情况。TIDC及SDC作为刺激细胞，脾脏T细胞作为反应细胞行混合淋巴细胞反应，同时加入B7 H1抗体或其对照抗体，XTT比色法检测T细胞增殖指数，ELISA法检测T细胞分泌IL 10的量。〖HT5W〗结果：〖HT5"SS〗B7 1及B7 2分子在TIDC表面的表达水平显著低于SDC（ P <0 0 1）；B7 H1分子在TIDC及SDC表面皆中度表达，表达水平无明显差异（ P >0.05）。TIDC刺激T细胞增殖能力显著低于SDC，且诱导T细胞分泌更多的IL 10。封闭DC表面B7 H1分子后，TIDC刺激T细胞增殖能力显著提高（ P <0.01），且诱导T细胞分泌IL 10的量明显减少（ P <0.01）；SDC刺激T细胞增殖能力及诱导T细胞分泌IL 10的量无明显变化（ P >0.05）。结论：封闭DC表面的B7 H1分子能显著提高TIDC活化T细胞的能力，可能解除TIDC介导的肿瘤免疫抑制

关键词：肿瘤浸润树突状细胞 T细胞 B7-H1 免疫功能

Effects of B7-H1 molecule blockade on tumor infiltrating dendritic cell mediated T cell function

Download Fulltext

CHEN Ya min LIANG Xiao hua HUANG Ruo fan ZHOU Xin li

Department of Oncology, Huashan Hospital, Shanghai Medical College, Fudan University, Shanghai 200032, China;Department of Oncology, Huashan Hospital, Shanghai Medical College, Fudan University, Shanghai 200032, China;Department of Oncology, Huashan Hospital, Shanghai Medical College, Fudan University, Shanghai 200032, China;Department of Oncology, Huashan Hospital, Shanghai Medical College, Fudan University, Shanghai 200032, China

Fund Project:Supported by the Scientific Research Foundation of Huashan Hospital Affiliated to Fudan University (No.145)

Abstract:

Objective: To explore the expression of B7 1, B7 2 and B7 H1 on tumor infiltrating dendritic cells (TIDC) and on splenic dendritic cells (SDC) , and to investigate TIDC mediated and SDC mediated T cell function after blocking B7 H1 expression in these dendritic cells. Methods: The TIDCs and SDCs were isolated from tumor bearing mice using anti mouse CD11c magnetic beads. The expression of B7 1, B7 2 and B7 H1 on TIDC and SDC was analyzed using flow cytometer. T cells were co cultured with TIDCs or SDCs for the mixed lymphocyte reaction (MLR), and monoclonal antibodies to B7 H1 or the isotype control antibodies were added to the MLR cultures. T cell proliferation was assessed using XTT method and the secretion of IL 10 was detected using ELISA. Results: B7 1 and B7 2 positive TIDCs were significantly less than SDCs (P <0.01). B7 H1 was moderately expressed on both TIDCs and SDCs (P >0.05). T cell proliferation stimulated by TIDCs was weaker than that stimulated by SDCs; T cells produced more IL 10 after TIDCs stimulation than after SDCs stimulation （ P <0.01）. After blocking B7 H1 on DCs, TIDCs showed a stronger stimulating ability on T cell proliferation compared with control antibodies, while SDCs did not have significant effect on T cell proliferation and production of IL 10. Conclusion: Blocking B7 H1 on TIDCs can significantly enhance their ability to activate T cells, and may elimilate TIDC mediated tumor immunosuppression.

Keywords:tumor infiltrating dendritic cell T cell B7-H1 immune function

查看全文 查看/发表评论 下载PDF阅读器

Copyright © Biother.Org™ All Rights Reserved; ISSN: 1007-385X CN 31-1725

主管单位：中国科学技术协会 主办单位：中国免疫学会、中国抗癌学会

地址：上海市杨浦区翔殷路800号 邮政编码：200433 京ICP备06011393号-2

本系统由北京勤云科技发展有限公司设计