



Jan 2, 2010

Dear Scientific Advisory Committee Members:

We are writing to invite you and your group to help with a unique type of information network that will be an important part of the new web based society in dendritic cell and vaccine science.

The society aims to fill two big gaps especially for younger scientists. One gap is a lack of funds to send students and postdoctoral fellows to meetings that bridge dendritic cell and vaccine science. A second gap is the insufficient interaction of two types of scientists, those studying the immune system especially dendritic cells, and those working on vaccines, including vaccines in areas where they do not currently exist, like cancer and allergy.

The latter is where we need your expertise as soon as possible. *We wish to invite your lab to contribute to the web site by joining its information team, e.g., once a month, provide one or more short summaries on subjects that are close to your research interest.* We hope that the web site will go live this month and that we will also, with your help when feasible, secure membership lists from various immunology societies worldwide to invite people to join.

Again, the idea is to contribute short summaries of 1-3 papers that bridge dendritic cell and vaccine science. These will appear continuously on the web site, from labs around the globe. In this way members can continually go to the web site not only to find out about meetings and jobs, but gain a vaccine science perspective with summaries from the literature. There are many journals and many areas of vaccine science, *including learning to suppress immunity in a specific and durable way.* So this sort of information center will be really helpful, especially if you report beyond the main journals. A sample follows from one of my lab members although she chose ones from major journals.

We think that the web site will make it attractive and valuable to be a member (\$US 50 in developed countries; \$500 for a lifetime membership) and thereby generate substantial income to fund travel for young scientists to meetings. *In other words, scientists world wide will help each other to bring immunology and vaccine science together and make it possible to get young people to meetings.*

We hope that it is possible for you to take part in this new endeavor, one which we are confident will reap continued and significant rewards. Initially, could you submit by e-mail to reviews@dc-vaccine.org or to my assistant Ms. Nulty, nultym@rockefeller.edu?

Best regards and have a wonderful new year,

for the executive committee



Vaccination against HPV-16 oncoproteins for vulvar intraepithelial neoplasia.

N Engl J Med 2009 Nov 5 361(19):1838-47

<http://www.ncbi.nlm.nih.gov/pubmed/19890126>

Vulvar intraepithelial neoplasia is a chronic premalignant disorder caused by high-risk types of human papillomavirus (HPV), with more than 75% involving HPV type 16 (HPV-16). Spontaneous regression is rare (<1.5%) and the recurrence rate after surgery is high. Now for the first time a therapeutic vaccine induces a convincing clinical response in a phase II study of HPV-16-associated, grade 3 vulvar intraepithelial neoplasia. The vaccine was composed of mixed synthetic long-peptides (30-40 amino acids in length) from the HPV-16 oncoproteins E6 and E7 administered in incomplete Freund's adjuvant or montanide. The vaccine was administered subcutaneously every 3 weeks. Prior research from this lab had shown that long peptides preferentially allow dendritic cells to cross-present peptides on MHC I. Adverse events were mild. 15 of 19 women (79%) had clinical responses and 9 of 19 patients (47%) had complete responses. The latter correlated with induction of HPV-16-specific T cell immunity. This study by Kenter et al provides evidence for the efficacy of a cancer therapeutic vaccine.

CD8(+) T lymphocyte mobilization to virus-infected tissue requires CD4(+) T-cell help.

Nature 2009 Nov 26 462(7272):510-3

<http://www.ncbi.nlm.nih.gov/pubmed/19898495>

CD4+ T cells are important players for priming and memory development of the cytotoxic CD8+ T lymphocyte (CTL) response. Now, Nakanishi et al. report a novel role for CD4+ T cells in CTL responses. Using a mouse model for herpes simplex type 2 (HSV-2) genital infection, the authors found that CD4+ T cells were required for the recruitment of CTL to viral-infected mucosal tissues. Mobilization of CTL was dependent on secretion of interferon- γ as well as chemokines (CXCL9/10) from CD4+ T cells. Strategies to promote CTL recruitment to infected or tumor tissue by manipulating CD4+ T cells should improve immunotherapy.

T helper 17 cells promote cytotoxic T cell activation in tumor immunity.

Immunity. 2009 Nov 20;31(5):787-98.

<http://www.ncbi.nlm.nih.gov/pubmed/19879162>

It is well known that T helper 1 cells provide help to anti-tumor CD8+ T cells. Orozco et al. present evidence that T helper 17 cells also can have a protective role in anti-tumor immunity. Initially, they noted that IL-17-deficient mice were more susceptible to development of B16-F10 melanoma. CD4+ T cells from OT-II mice were then polarized to Th17 cells *in vitro* with cytokine cocktail. Transfer of OVA-specific Th17 cells intravenously together with CD8+ T cells from OT-I mice enhanced the CD8 T cell responses as measured by IFN- γ production and proliferation ability. Th17 transfer also helps CD8 T cells reject established B16-OVA tumor.