

THE MIDWINTER CONFERENCE OF IMMUNOLOGISTS  
POSTER ABSTRACT - 2005

Name:     Louise McHeyzer-Williams     E-mail :     louisemw@scripps.edu      
Use same name on subject line of e-mail when transmitting abstract, not "Asilomar abstract."

In the box provided below, briefly summarize the theme of your abstract. By Friday, December 17, 2004, send an electronic copy and a hard copy with this signed form to Dr. Carl F. Ware, Division Molecular Immunology, La Jolla Institute for Allergy and Immunology, 10355 Science Center Drive, San Diego, CA 92121.

E-mail: [carl\\_ware@lai.org](mailto:carl_ware@lai.org).

All abstracts are accepted for poster presentation. Receipt of your abstract will not be confirmed.  
(Poster size: 4'w x 4'h, maximum)

Do you approve that this abstract appears on the MCI web page? YES ( X ) NO ( )  
E-mail your abstract as requested above.  
Send this form with hard copy of abstract. Sign: \_\_\_\_\_ Date: \_\_\_\_\_

### **Development of Antigen-Specific B Cell Memory**

Louise J. McHeyzer-Williams, Adam P. O'Connor & Michael. G. McHeyzer-Williams  
The Scripps Research Institute, La Jolla, CA

We have recently demonstrated the presence of two distinct subsets of antigen-specific memory B cells following immunization with thymus dependent (TD) antigens. These memory subsets are phenotypically separable as antibody isotype-switched, antigen-binding B cells that express CD79b (I $\mu$ B BCR co-receptor) with differential expression of a glycosylation variant of CD45 that is detected by the binding of mAb 6B2. Both 6B2+ and 6B2- non-secreting memory B cell subsets lack the expression of Syndecan-1 (CD138) found on all sub-types antibody-secreting cells in TD B cell responses. In this current study, we deepen molecular analyses of the memory subsets with emphasis on developmental programming and differential function in vivo. Single cell RT-PCR analysis identifies somatically mutated I $\mu$  in 45-50% of either memory B cell subset, strongly arguing against non-B cell contamination. Gene expression directly ex vivo and Q-PCR analysis confirms the presence and levels of I $\mu$  and CD79b in each memory B cell subset. Further phenotypic and genotypic analysis helps to extend our understanding of the unique attributes of each memory sub-population and guides our studies on the mechanism of long-term survival in vivo. Differential expression of CD40 and capacity to respond to CD40 ligation in vitro highlights the distinct re-activation requirements of each memory B cell subset in vivo. Hence, these two memory B cell subsets express unique developmental programs that control subsequent cell fate and impact memory B cell function upon antigen recall.