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Meeting abstract

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Resting and activated states of the B cell antigen receptor

J Yang*, Y Kulathu, S Infantino, D Medgyesi and M Reth

Address: Centre of biological signalling studies, bioss, University of Freiburg, and Dept. of Mol. Immunol, MPI for Immunobiology, Freiburg, Germany

* Corresponding author

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Each B cell contains up to 100,000 B cell antigen receptor (BCR) complexes on its surface, which only become fully active on exposure of the B cell to its cognate antigen. Most studies on BCR function aims at a better understanding how the BCR becomes active and transmits its signal to the many signaling pathways inside the cell. Another, albeit related question, is how the B cells ensure that the many BCR complexes stay (in most cases) in a silent inert conformation. This problem of activation control the BCR shares with many of its signaling elements, be it kinases, phosphatases or adaptors, which all have to be regulated tightly in their localization and activity. For the later element autoinhibition plays an essential role in this regulation. In a biochemical study we previously have found that on resting B cells the BCR resides in oligomeric and monomeric forms. We now have conducted experiments that suggest that the BCR also can resume an autoinhibitory conformation and that BCR oligomerization plays an important role in this process. Specifically oligomerization of the BCR in conjunction with the kinase-phosphatase equilibrium at the BCR contributes to the tight control of BCR activation. Our studies resulted in a new model of the resting BCR and its activation.

We also study how the cytosolic protein tyrosine kinase Syk is regulated and activated by the BCR. Syk is also regulated by auto-inhibition and only becomes fully active when recruited to the BCR. We now have identified Syk mutant that are altered in auto-inhibition and tested their activity in the S2 Schneider cell reconstitution system as well as in reconstituted Syk deficient B cell lines. Our data show that a tight Syk regulation is essential for normal B-lymphocytes development.