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Negative regulatory role of the actin cytoskeleton in B cell activation and self tolerance

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The actin cytoskeleton is critical for both signal activation and antigen processing in B cells. Actin promotes the activation of B cell receptor (BCR) through controlling B cell spreading and BCR aggregation. Our recent studies found that in addition to signaling activation, the actin cytoskeleton plays a critical role in the attenuation of BCR signaling. Actin regulates signaling attenuation by controlling actin accumulation at the B cell surface, switching B cell spreading to contraction, and driving the merger of BCR aggregates into a polarized central cluster. Defects in these cellular processes by deleting genes of specific actin regulators from mice lead to increased BCR signaling, delayed signaling attenuation, and decreased self tolerance of B cells. These results suggest that actin regulators are potential targets for manipulating B cell-mediated autoimmunity.

Biography

Song completed her Ph.D. in cell biology at the Kansas State University and had postdoctoral trainings at University of California, San Francisco and Northwestern University. She joined the faculty at University of Maryland in 1996 where she has directed research on cell biology of B lymphocytes. Her laboratory focuses on the activation mechanisms of B cells. She has published numerous papers and served on editorial boards of several journals and grant panels.

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