This week in therapeutics

Indication	Target/marker/pathway	Summary	Licensing status	Publication and contact information
Cancer				
Cancer	Neuropilin 1 (NRP1); semaphorin 4A (SEMA4A)	<p>Mouse studies suggest blocking SEMA4A-NRP1 signaling could increase an antitumor immune response without causing pathogenic autoimmunity. In mice with T_{reg}-specific <i>Nrp1</i> knockout, injection of cancer cell lines led to less tumor growth and greater antitumor immune responses than what was seen in mice with intact <i>Nrp1</i>. Mice with deletion of <i>Nrp1</i> in T_{reg} cells did not develop autoimmunity, and <i>Nrp1</i>-deleted T_{reg} cells could maintain immune system homeostasis. In an ELISA, SEMA4A was shown to interact directly with NRP1. In mouse models of cancer, injection of SEMA4A- or NRP1-targeted antibodies decreased tumor growth compared with injection of control antibodies. Next steps include determining if the SEMA4A-NRP1 signaling axis in T_{reg} cells is upregulated in human solid tumors.</p> <p>SciBX 6(32); doi:10.1038/scibx.2013.854 Published online Aug. 22, 2013</p>	<p>Patent application filed; available for licensing from St. Jude Children's Research Hospital</p> <p>Contact: Scott Elmer, St. Jude Children's Research Hospital, Memphis, Tenn. e-mail: scott.elmer@stjude.org</p>	<p>Delgoffe, G.M. <i>et al. Nature</i>; published online Aug. 4, 2013; doi:10.1038/nature12428</p> <p>Contact: Dario A.A. Vignali, St. Jude Children's Research Hospital, Memphis, Tenn. e-mail: vignali.lab@stjude.org</p>