This week in techniques

Approach	Summary	Licensing status	Publication and contact information
Drug delivery			
<i>Ex vivo</i> GI organoids to model cancer progression and evaluate driver genes	<p><i>Ex vivo</i> and mouse studies suggest GI organoid cultures could help model colorectal cancer (CRC). Primary GI organoids were produced by growing mouse GI explants containing epithelial and mesenchymal components in an air-liquid interface culture system. In primary GI organoids, combinations of <i>adenomatous polyposis coli</i> (<i>Apc</i>) knockout, mutant <i>K-Ras</i> (<i>Kras</i>) expression, <i>p53</i> knockdown or <i>SMAD family member 4</i> (<i>Madh4</i>; <i>Smad4</i>; <i>Dpc4</i>) knockdown led to oncogenic transformation that recapitulated histological human CRC subtypes. All four modifications together resulted in the most severe colorectal adenocarcinoma phenotype. In <i>Apc</i> knockout organoids, <i>miR-483</i> overexpression was sufficient to cause dysplasia in culture and tumor formation in mice after subcutaneous transplantation, suggesting <i>miR-483</i> acts as a driver gene for CRC. Next steps include evaluating function and temporal requirements of additional driver gene candidates and extending the findings to human organoids.</p> <p>SciBX 7(25); doi:10.1038/scibx.2014.747 Published online June 26, 2014</p>	Patent and licensing status undisclosed	<p>Li, X. <i>et al. Nat. Med.</i>; published online May 25, 2014; doi:10.1038/nm.3585 Contact: Calvin J. Kuo, Stanford University School of Medicine, Stanford, Calif. e-mail: cjkuo@stanford.edu</p>