

## This week in therapeutics

| Indication    | Target/marker/pathway  | Summary   | Licensing status                            | Publication and contact information  |
|---------------|--|---|---|--|
| <b>Cancer</b> |  |   |   |  |
| Brain cancer  | H3 histone family 3A (H3.3A; H3F3A); v-myc myelocytomatosis viral related oncogene neuroblastoma derived (MYCN; NMYC); checkpoint kinase 1 (Chk1); aurora kinase A (AURKA; Aurora-A) | <p>Cell culture studies suggest inhibiting Chk1 or AURKA may help treat pediatric and young adult patients with <i>H3F3A</i>-driven glioblastoma. In a glioblastoma cell line with G34-mutant <i>H3F3A</i>, <i>MYCN</i> was identified as the most highly overexpressed gene, and Chk1 and AURKA were identified as regulators of cell proliferation. In the same cell line, an AURKA inhibitor dose-dependently decreased <i>MYCN</i> levels and cell viability compared with no treatment. Next steps include testing AURKA and Chk1 inhibitors in a larger number of glioblastoma cell lines and building <i>in vivo</i> models to test the effects of the inhibitors in orthotopic brain tumors.</p> <p>At least eight companies have AURKA inhibitors in preclinical to Phase III testing in various cancers.</p> <p>At least six companies have Chk1 inhibitors in preclinical to Phase II trials in various cancer indications.</p> <p><b>SciBX 6(18); doi:10.1038/scibx.2013.433</b><br/> <b>Published online May 9, 2013</b></p> | Unpatented; licensing status not applicable | <p>Bjerke, L. <i>et al. Cancer Discov.</i>; published online March 28, 2013; doi:10.1158/2159-8290.CD-12-0426</p> <p><b>Contact:</b> Chris Jones, The Institute of Cancer Research, Surrey, U.K.<br/> e-mail: <a href="mailto:chris.jones@icr.ac.uk">chris.jones@icr.ac.uk</a></p> |