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See the screen shots on the last page for an example.

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Questions about the CCR Portal should be directed to Ave Cline (x5176 or clineav@mail.nih.gov) or Sue Fox (x1923 or foxs@mail.nih.gov).

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For access to this system please contact Tammy Eyler (x5271 or Tammy.Eyler@nih.gov).

Thanks to Sue Fox, CCR Office of Communications, and to Tammy Eyler, Basic Science Program, Leidos Biomedical Research, Inc. for their contributions to this document.

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Effective March 4, 2014, ACS will provide authors with a complimentary ACS AuthorChoice+12 license for each article published in 2014 that acknowledges funding from NIH. The AuthorChoice+12 license provides open availability of ACS copyrighted articles on the Web 12 months after first online publication by ACS. ACS will deposit the article with the NIHMS at no charge. This will only be effective for 2014.

Starting in 2015, ACS will offer authors two options to submit their articles from ACS publications to the NIHMS:

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- **Option 2** – The **author** may **self-deposit for free** the **peer-reviewed manuscript, accepted for publication but prior to ACS' copy editing and production**, with NIH, for open availability **12 months after publication**.

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- **NCI at Frederick Scientific Library**—call or email your questions to x1093 or NCIFredLibrary@mail.nih.gov. We can also come to your desk to help you.

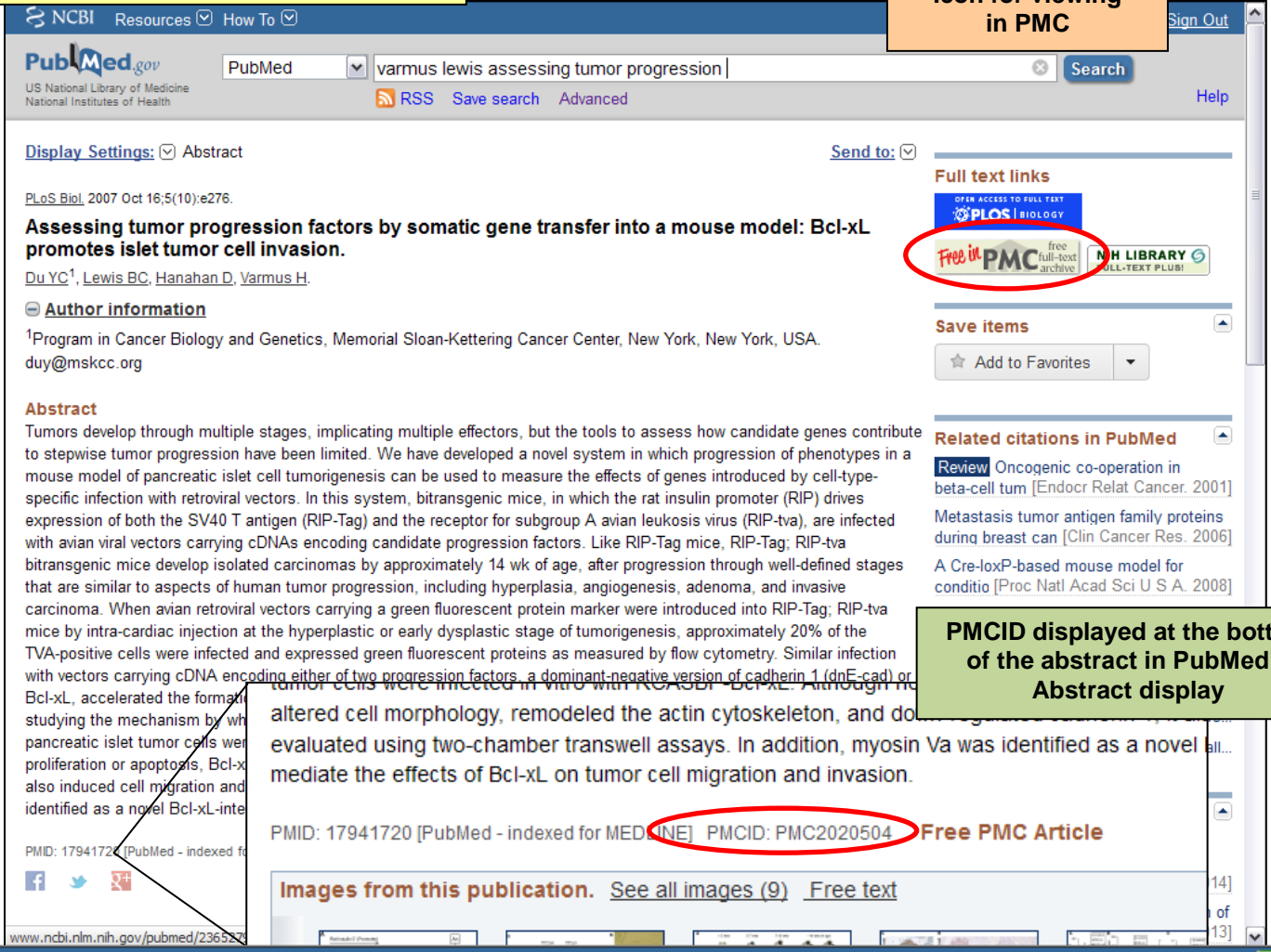
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Assessing tumor progression factors by somatic gene transfer into a mouse model: Bcl-xL promotes islet tumor cell invasion.

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Abstract

Tumors develop through multiple stages, implicating multiple effectors, but the tools to assess how candidate genes contribute to stepwise tumor progression have been limited. We have developed a novel system in which progression of phenotypes in a mouse model of pancreatic islet cell tumorigenesis can be used to measure the effects of genes introduced by cell-type-specific infection with retroviral vectors. In this system, bitransgenic mice, in which the rat insulin promoter (RIP) drives expression of both the SV40 T antigen (RIP-Tag) and the receptor for subgroup A avian leukosis virus (RIP-tva), are infected with avian viral vectors carrying cDNAs encoding candidate progression factors. Like RIP-Tag mice, RIP-Tag; RIP-tva bitransgenic mice develop isolated carcinomas by approximately 14 wk of age, after progression through well-defined stages that are similar to aspects of human tumor progression, including hyperplasia, angiogenesis, adenoma, and invasive carcinoma. When avian retroviral vectors carrying a green fluorescent protein marker were introduced into RIP-Tag; RIP-tva mice by intra-cardiac injection at the hyperplastic or early dysplastic stage of tumorigenesis, approximately 20% of the TVA-positive cells were infected and expressed green fluorescent proteins as measured by flow cytometry. Similar infection with vectors carrying cDNA encoding either of two progression factors, a dominant-negative version of cadherin 1 (dnE-cad) or Bcl-xL, accelerated the formation of carcinomas. In addition, dnE-cad and Bcl-xL altered cell morphology, remodeled the actin cytoskeleton, and down-regulated myosin Va. These effects were evaluated using two-chamber transwell assays. In addition, myosin Va was identified as a novel effector that mediates the effects of Bcl-xL on tumor cell migration and invasion.

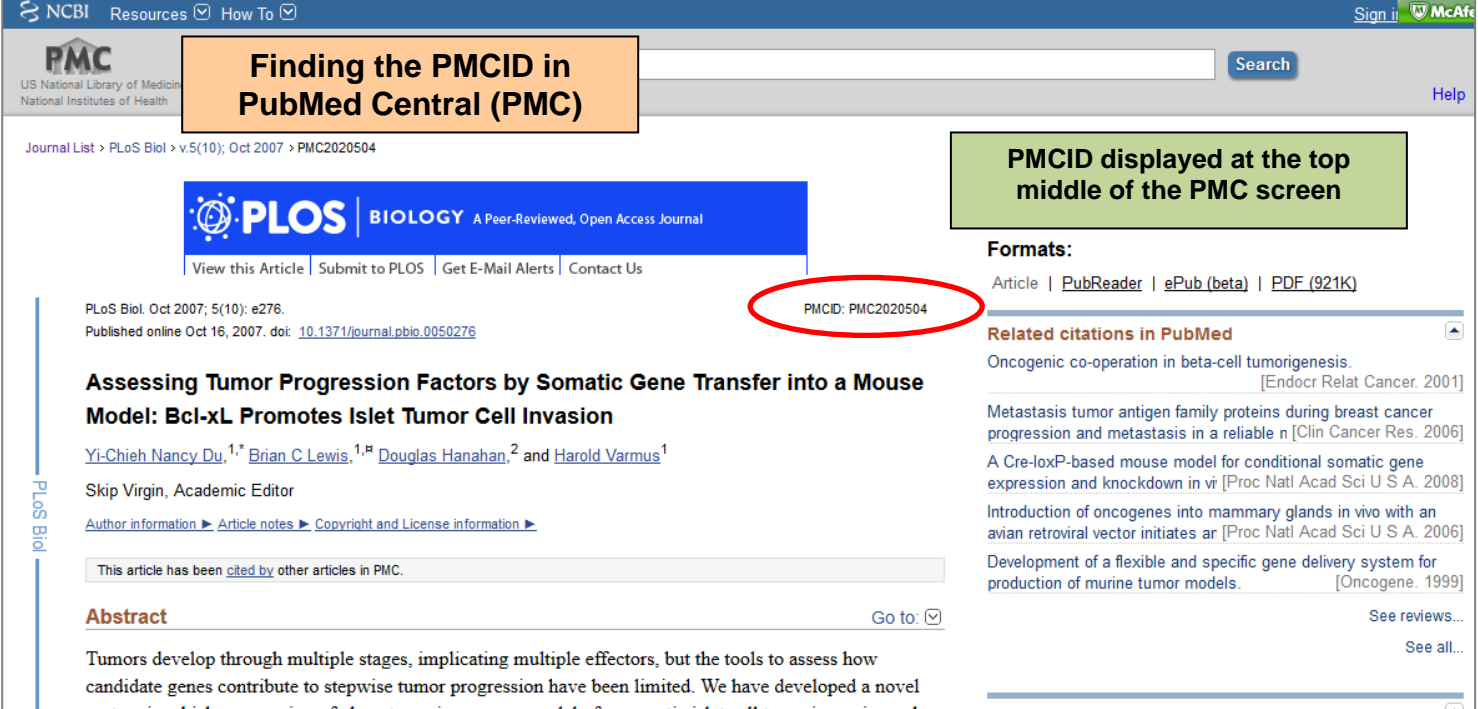
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Assessing Tumor Progression Factors by Somatic Gene Transfer into a Mouse Model: Bcl-xL Promotes Islet Tumor Cell Invasion

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Abstract

Tumors develop through multiple stages, implicating multiple effectors, but the tools to assess how candidate genes contribute to stepwise tumor progression have been limited. We have developed a novel system in which progression of phenotypes in a mouse model of pancreatic islet cell tumorigenesis can be used to measure the effects of genes introduced by cell-type-specific infection with retroviral vectors. In this system, bitransgenic mice, in which the rat insulin promoter (RIP) drives expression of both the SV40 T antigen (RIP-Tag) and the receptor for subgroup A avian leukosis virus (RIP-tva), are infected with avian viral vectors carrying cDNAs encoding candidate progression factors. Like RIP-Tag mice, RIP-Tag; RIP-tva bitransgenic mice develop isolated carcinomas by approximately 14 wk of age, after progression through well-defined stages that are similar to aspects of human tumor progression, including hyperplasia, angiogenesis, adenoma, and invasive carcinoma. When avian retroviral vectors carrying a green fluorescent protein marker were introduced into RIP-Tag; RIP-tva mice by intra-cardiac injection at the hyperplastic or early dysplastic stage of tumorigenesis, approximately 20% of the TVA-positive cells were infected and expressed green fluorescent proteins as measured by flow cytometry. Similar infection with vectors carrying cDNA encoding either of two progression factors, a dominant-negative version of cadherin 1 (dnE-cad) or Bcl-xL, accelerated the formation of carcinomas. In addition, dnE-cad and Bcl-xL altered cell morphology, remodeled the actin cytoskeleton, and down-regulated myosin Va. These effects were evaluated using two-chamber transwell assays. In addition, myosin Va was identified as a novel effector that mediates the effects of Bcl-xL on tumor cell migration and invasion.

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