

Supplementary

Activation of PDGFr- β Signaling Pathway after Imatinib and Radioimmunotherapy Treatment in Experimental Pancreatic Cancer

Michio Abe ¹, Zbigniew P. Kortylewicz ², Charles A. Enke ², Elizabeth Mack ²
and Janina Baranowska-Kortylewicz ^{2,*}

¹ Minamata City Hospital and Medical Center, Minamata City, Kumamoto 867, Japan;
E-Mail: PFG02651@nifty.ne.jp

² Department of Radiation Oncology, J. Bruce Henriksen Cancer Research Laboratories, University of Nebraska Medical Center, Omaha, NE 68198, USA; E-Mails: zkortylewicz@unmc.edu (Z.P.K.); cenke@unmc.edu (C.A.E.); elizabeth.mack@unmc.edu (E.M.)

* Author to whom correspondence should be addressed; E-Mail: jbaranow@unmc.edu;
Tel.: +1-402-559-8906; Fax: +1-402-559-9127.

Figure S1. Sections of pancreatic human adenocarcinoma SW1990 xenografts grown subcutaneously in athymic mice given only PBS (controls, upper row) or treated with imatinib (lower row). Sections were cut from several xenografts to illustrate the tissue heterogeneity. The structure of these tumors and their heterogeneity is best viewed when the page size is at 500%. H&E, original magnification $\times 10$.

