NEOPLASIA
www.neoplasia.com
Volume 14 Number 12 December 2012 pp. 1278–1289 1278

Overcoming Intratumor Heterogeneity of Polygenic Cancer Drug Resistance with Improved Biomarker Integration[1]

Alnawaz Rehemtulla

Departments of Radiation Oncology and Radiology, University of Michigan, Ann Arbor, MI

Abstract

Improvements in technology and resources are helping to advance our understanding of cancer-initiating events as well as factors involved with tumor progression, adaptation, and evasion of therapy. Tumors are well known to contain diverse cell populations and intratumor heterogeneity affords neoplasms with a diverse set of biologic characteristics that can be used to evolve and adapt. Intratumor heterogeneity has emerged as a major hindrance to improving cancer patient care. Polygenic cancer drug resistance necessitates reconsidering drug designs to include polypharmacology in pursuit of novel combinatorial agents having multitarget activity to overcome the diverse and compensatory signaling pathways in which cancer cells use to survive and evade therapy. Advances will require integration of different biomarkers such as genomics and imaging to provide for more adequate elucidation of the spatially varying location, type, and extent of diverse intratumor signaling molecules to provide for a rationale-based personalized cancer medicine strategy.

Neoplasia (2012) 14, 1278–1289

Introduction

In Sun Tzu's book *The Art of War*, method and discipline are part of the five constant factors governing one's deliberations, which are considered necessary to achieve success. In other words, proper assessment of the strengths and weaknesses of your capabilities and those of your opponent are crucial before engagement. For many centuries, surgery was the only treatment option available for cancer patients. Advancements in anesthesia, antiseptic practices, blood transfusions, and antibiotics were landmark achievements providing for the field of "surgical oncology" to develop. However, during this time the observation of cancer cell spread or metastasis necessitated additional developments including radiation therapy and chemotherapy. Initially, the use of drugs involved cytotoxic chemotherapy for killing rapidly proliferating cancer cells, which evolved to combination therapy using drugs with different molecular mechanisms and toxicity profiles. It has long been rationalized that the ability to eliminate a tumor required identification of a druggable target presented by the tumor, which could be exploited as a differentiable vulnerability (either by presence or amount) apart from the host organism.

Attack by a Stratagem

The exquisite scientific progress achieved over the past few decades have had a major impact in improving our understanding of tumor treatment sensitivity and response to therapeutic interventions. In the current post-genomic era, a clearer picture is emerging revealing an ever increasing complex array of interconnected adaptable signaling pathways on which defined pathogenic driver mutations emerge and on which tumor cells become addicted to for survival. This understanding led to the establishment of a new generation of cancer drugs termed molecularly targeted cancer therapies developed to block the growth and spread of cancer through interfering with specific signaling molecules involved in tumor growth and progression. Molecularly targeted agents offered the ability to potentially treat genetically defined subgroups of patients by focusing on cellular and molecular alterations specific to their individual cancer. Major clinical advances were anticipated to be achieved through the development and use of targeted agents based on the belief that targeted cancer therapies would be more effective than traditional chemotherapy and radiotherapy. It was also hoped that targeted cancer agents would provide additional

Address all correspondence to: Alnawaz Rehemtulla, PhD, University of Michigan School of Medicine, 109 Zina Pitcher Place, Biomedical Sciences Research Building, Ann Arbor, MI 48109-2200. E-mail: alnawaz@umich.edu
[1]Grant funding from NIH P01CA085878 and P50CA093990.
Received 11 December 2012; Revised 11 December 2012; Accepted 11 December 2012

DOI 10.1593/neo.122096

benefits because while being less harmful to normal cells, systemic side effects would be if not ameliorated altogether, at least minimized, thus improving a patient's overall quality of life. Furthermore, individualization of cancer patient care was now seen as possible, as confirmation of the presence of a particular target within a tumor could be achieved in a relatively straightforward manner through molecular genomic analysis of an individual patient's tumor using a biopsy sample obtained from a patient's tumor. By matching the drug to the tumor target, major clinical successes seemed to be well within reach.

Variation in Tactics

Experience has now showed that targeted therapies have limitations that include potential for tumor cells to develop resistance. For example, some patients have developed resistance to imatinib (Gleevec) through a mutation in the *BCR-ABL* gene that alters the shape of the protein so that its affinity for drug binding is reduced, decreasing treatment efficacy. In fact, a wide variety of tumor cell adaptations in signaling molecules may occur following exposure to targeted agents, providing the cells with an opportunity to gain the upper hand through acquired resistance, thus escaping elimination. Typically, alternative targeted therapies that could possibly overcome the acquired resistance are not available nor are the specific cellular adaptations that occurred easily determined in real time. Owing to these issues in part, targeted therapies are being used in combination with other targeted therapies or with traditional chemotherapy and radiation therapy to provide for a more aggressive treatment approach.

What was described thus far was an overall approach to cancer treatment from the perspective of an individual tumor mass having a single or major oncogenic driver that can be targeted following identification of the target through analysis of a biopsy sample. However, we now know that the composition of tumors can be comprised of many different cell subpopulations, each which can harbor different genotypic and phenotypic characteristics leading to a diversity of biologies. This knowledge can be very disconcerting, leaving one with the feeling of "two steps forward and one step backwards" in the war on cancer. The impact associated with the relatively recent understanding of the presence of significant intratumor heterogeneity on tumor therapy cannot be underestimated. Multiple cellular subpopulations with different genetic and phenotypic characteristics along with their associated tremendous three-dimensional spatial variation within a single lesion portends the fact that a specific lesion does not have a single target but rather multiple oncogenic targets that must be overcome to achieve optimized therapeutic benefit [Yap TA (2012). *Sci Transl Med* **4**, 127].

Terrain

There are potentially a myriad of factors that can contribute to intratumor heterogeneity. For example, heterogeneous cellular microenvironments within a tumor mass can act to drive regional phenotypic evolution and diversity with associated genetic mutations. For example, spatially distinct regions within a tumor with limited blood flow would have low oxygenation levels that could lead to tumor cells adapted to a microenvironment of acidosis and hypoxia resulting in augmented survival pathways and enhanced resistance to chemoradiation. Moreover, genetic instability within a tumor cell population provides opportunities for the emergence of spatially varying cells expressing alternate oncogenes and tumor suppressor genes further complicating the development of an adequate treatment strategy for an individual tumor. Recent findings have in fact revealed significant intratumor heterogeneity, and branched evolution was present in primary renal carcinomas and associated metastatic sites using multiregion sequencing [Gerlinger M (2012). *N Engl J Med* **366**, 892]. In this study, multiregion genetic analysis of four different tumors revealed that intratumor heterogeneity was present in every tumor analyzed. Tumors were found to have spatially separated heterogeneous somatic mutations, patterns of allelic imbalance, and chromosomal imbalances that led to phenotypic intratumor diversity and uniformity. It was also shown that a single tumor-biopsy specimen only revealed a minority of genetic aberrations (including mutations, allelic imbalance, and ploidy) that were actually present in the tumor mass. Thus, characterization of intratumor heterogeneity using next-generation sequencing necessitates our rethinking of how best to approach the development of effective personalized medicine, which, until now, has relied on single biopsy samples to delineate the mutational landscape of a patient's cancer. It is clear that genomic analyses using single tumor-biopsy specimens underestimate the overall mutational burden of tumors because the presence of significant intratumor heterogeneity requires the issue of sampling bias to be addressed to achieve improved outcome.

Intratumor heterogeneity of protein function may nurture tumor adaptation through a Darwinian selection process leading to improper selection of drugs resulting in an ineffective and thus disastrous treatment outcome. Due to limitations in information obtainable from single tumor-biopsy samples, the presence of intratumor heterogeneity is anticipated to provide abridged information related to the true tumor genomic landscape resulting in unending challenges to development of personalized medicine for cancer patients. Due to the presence of intratumor heterogeneity, intrinsic and acquired resistance to cytotoxic chemotherapy, ionizing radiation, and molecularly targeted drugs is an unavoidable consequence including the need to target multiple oncogenic drivers of the tumor. One proposed solution is to use the information related to polygenic drug resistance to develop a rationally designed approach using combinatorial targeted therapy to address the heterogeneity of targets disseminated throughout the tumor mass. One of the key issues related to combination therapies is the potential for severe and undesirable side effects. In an effort to minimize these toxicities, new approaches to drug development are incorporating simultaneous targeting moieties within a single structurally designed molecule [Guerrant W (2012). *J Med Chem* **55**, 1477]. Development of novel multivalent ligands to modulate several cancer targets simultaneously may provide a useful approach for combating the inherent intratumor heterogeneous diversity of protein function encountered in clinical experience.

The Use of Spies

The concept of significant spatially varying somatic mutations along with the need to delineate the major contributors of such within an individual's tumor necessitates the need for obtaining additional multiregional information to optimize therapy and to predict treatment outcome. Obtaining multiple biopsies may be problematic especially in patients with multifocal metastatic disease and following treatment wherein a tumor may adapt and thus gain resistance to the intervention in real time. An alternate proposed strategy is to use state-of-the-art clinical imaging modalities to complement the information obtained from genetic analysis to further characterize tumor spatial heterogeneity [Basu S (2011). *Eur J Nucl Med Mol Imaging* **38**, 991]. Imaging can be used to reveal intratumor phenotypic heterogeneity that likely reflects the underlying and spatially corresponding genetic

Table 1. Summary of Published Articles.

Subject	2010	2011	2012
Cancer genetics	[14,37,45,51,64,67,69–71,90,92,93,100,104]	[123,137,139,143,146,160,164,180,205,206,209]	[231,232,235,238,239,250,258,261,263,268,269, 273,278,283,289,294,299,303,320,323]
Cell and tumor biology	[1,2,5,8,10,15–17,19,21,22,30–33,35,36, 40,41,44,48,52,53,55,56,58,59,61–63, 65,66,73,74,79–81,83,85,86,89,91, 94–96,98,99,101,103,105]	[107–109,115–117,124,130,132–136, 141,145,151,152,154,156,161,163, 168,171,173,174,176,181,182, 188,191–197,199,201,208,210–214]	[216–220,222,224,227,228,233,234,236,237,244, 248,249,256,257,260,262,267,271,275,279, 282,284,286,290–292,301,302,304–306, 313–317,319]
Experimental therapeutics	[4,6,7,11,20,27,28,38,46,50,54,60,87,88,106]	[112,114,119–122,127,129,138,140, 142,147–149,167,172,175,184–186, 189,190,202–204,207,215]	[221,225,226,240,242,246,253,254,259,266,270, 274,276,280,281,287,288,293,296,297,300, 308–310,312,321,322,324]
Tumor immunology	[13,29,43,57,68,78,97]	[110,113,159,166,170,178,179,187, 198,200]	[229,230,245,251,277,285,307,318]
Epidemiology and prevention	[39]		
Cancer imaging	[25,75,84]	[118,128,131,150,153,165,183]	[247,252,255,264,265]
Clinical investigations	[3,9,12,23,24,26,34,42,47,49,77,82,102]	[125,169]	[223,243]
Animal models	[18,76]	[111,126,144,177]	[241,311]

diversity. For example, positron emission tomography, magnetic resonance imaging (MRI), and computed tomography (CT) all provide multislice high-resolution anatomic as well as functional images that can be repeated over time before, during, and following treatment intervention. These imaging modalities are well suited for interrogating the three-dimensional landscape of tumors noninvasively and over time. While traditional clinical assessment of tumor images is conducted by a trained radiologist, the complexities and subtleties of spatially varying signal intensities may be difficult to extract and interpret by the naked eye. Traditionally, whole-tumor statistical averages (histograms) of quantified imaging metrics have been used to characterize tumors before and following treatment. However, this type of analysis relegates the spatial information to a whole-tumor average measurement. More recently, emergence of a voxel-by-voxel–based approach for quantifying changes over time spatially has been found to provide more sensitivity for detecting changes over time during therapy as well as retaining the spatial information within the anatomic context of the tumor [Galban CJ (2011). *Clin Cancer Res* **17**, 4760]. The voxel-based method has been applied across imaging modalities and provides new opportunities to assess intratumor heterogeneity [Laymon CM (2012). *Magn Reson Imaging* **30**, 1278]. Additional approaches for assessment of tumor heterogeneity using digital image post-processing algorithms include texture analysis methods [Davnall F (2012). *Insights Imaging*].

Imaging can provide significant and timely clinical patient management support, as it can provide rapid and objective assessment of tumor therapeutic response. However, response to therapeutic intervention may be initially successful, but rapid emergence of spatially varying resistance is likely and development of a noninvasive imaging approach with the ability to detect this eventuality in a timely manner would allow for rapid deployment of alternate interventions that would be anticipated to optimize therapeutic outcome. Diffusion-weighted MRI has been shown to provide a sensitive measure of tumor response throughout the course of treatment. In addition, diffusion-weighted MRI was reported to be sensitive enough to detect real-time emergence of resistance in an animal tumor model [Lee KC (2006). *Cancer Res* **66**, 4692]. The overall ability of imaging metrics to delineate spatially varying and treatment-associated changes in tumor structure and function provides complementary information to biopsy-derived molecular genetic information related to tumor target presentation and will ultimately improve drug selection and patient care.

Summary

As described above, genotypic and phenotypic diversity has a tremendous impact on cancer growth and intervention. The journal *Neoplasia* provides peer-reviewed diverse information related to basic cancer biology, as it relates to clinical relevance and serves as a rapid conduit to disseminate relevant and timely information to the international community. Exemplifying the significant diversity of articles published in *Neoplasia* over the past 3 years, articles that cover areas of cancer research including cell and tumor biology, imaging, genetics, experimental therapeutics, and clinical investigations are published (Table 1). The readership of *Neoplasia* is provided with broad-based state-of-the-art information comprising many key pieces of the overall oncology puzzle. Furthermore, the immediate availability of *Neoplasia* articles to the world-wide clinical cancer research community is a key feature of *Neoplasia*, allowing authors' research findings to be made available to the largest possible readership ensuring that published articles will have a significant impact. Finally, as the Editor in Chief and, along with the Editorial Board, we have been pleased with the overall success that this effort has in serving the cancer research community. We thank the scientific groups who have entrusted the dissemination of their research findings to our publication and we look forward to continued progress in knowledge, which will be used to improve cancer patient care and outcome.

References

[1] Aleshin A and Finn RS (2010). SRC: a century of science brought to the clinic. *Neoplasia* **12**, 599–607.

[2] Andl CD, McCowan KM, Allison GL, and Rustgi AK (2010). Cathepsin B is the driving force of esophageal cell invasion in a fibroblast-dependent manner. *Neoplasia* **12**, 485–498.

[3] Arum CJ, Anderssen E, Viset T, Kodama Y, Lundgren S, Chen D, and Zhao CM (2010). Cancer immunoediting from immunosurveillance to tumor escape in microvillus-formed niche: a study of syngeneic orthotopic rat bladder cancer model in comparison with human bladder cancer. *Neoplasia* **12**, 434–442.

[4] Bassi DE, Zhang J, Cenna J, Litwin S, Cukierman E, and Klein-Szanto AJ (2010). Proprotein convertase inhibition results in decreased skin cell proliferation, tumorigenesis, and metastasis. *Neoplasia* **12**, 516–526.

[5] Belaguli NS, Aftab M, Rigi M, Zhang M, Albo D, and Berger DH (2010). GATA6 promotes colon cancer cell invasion by regulating urokinase plasminogen activator gene expression. *Neoplasia* **12**, 856–865.

[6] Bera S, Greiner S, Choudhury A, Dispenzieri A, Spitz DR, Russell SJ, and Goel A (2010). Dexamethasone-induced oxidative stress enhances myeloma cell radiosensitization while sparing normal bone marrow hematopoiesis. *Neoplasia* **12**, 980–992.

[7] Brauer R, Wang LC, Woon ST, Bridewell DJ, Henare K, Malinger D, Palmer BD, Vogel SN, Kieda C, Tijono SM, et al. (2010). Labeling of oxidizable proteins with a photoactivatable analog of the antitumor agent DMXAA: evidence for redox signaling in its mode of action. *Neoplasia* **12**, 755–765.

[8] Brioschi M, Fischer J, Cairoli R, Rossetti S, Pezzetti L, Nichelatti M, Turrini M, Corlazzoli F, Scarpati B, Morra E, et al. (2010). Down-regulation of microRNAs 222/221 in acute myelogenous leukemia with deranged core-binding factor subunits. *Neoplasia* **12**, 866–876.

[9] Broderick SR, Golas BJ, Pham D, Towe CW, Talbot SG, Kaufman A, Bains S, Huryn LA, Yonekawa Y, Carlson D, et al. (2010). SCCRO promotes glioma formation and malignant progression in mice. *Neoplasia* **12**, 476–484.

[10] Brown JE and Sim S (2010). Evolving role of bone biomarkers in castration-resistant prostate cancer. *Neoplasia* **12**, 685–696.

[11] Burden-Gulley SM, Gates TJ, Burgoyne AM, Cutter JL, Lodowski DT, Robinson S, Sloan AE, Miller RH, Basilion JP, and Brady-Kalnay SM (2010). A novel molecular diagnostic of glioblastomas: detection of an extracellular fragment of protein tyrosine phosphatase μ. *Neoplasia* **12**, 305–316.

[12] Campbell NE, Greenaway J, Henkin J, Moorehead RA, and Petrik J (2010). The thrombospondin-1 mimetic ABT-510 increases the uptake and effectiveness of cisplatin and paclitaxel in a mouse model of epithelial ovarian cancer. *Neoplasia* **12**, 275–283.

[13] Choudhary R, Li H, Winn RA, Sorenson AL, Weiser-Evans MC, and Nemenoff RA (2010). Peroxisome proliferator–activated receptor-γ inhibits transformed growth of non–small cell lung cancer cells through selective suppression of Snail. *Neoplasia* **12**, 224–234.

[14] Clauss A, Ng V, Liu J, Piao H, Russo M, Vena N, Sheng Q, Hirsch MS, Bonome T, Matulonis U, et al. (2010). Overexpression of elafin in ovarian carcinoma is driven by genomic gains and activation of the nuclear factor κB pathway and is associated with poor overall survival. *Neoplasia* **12**, 161–172.

[15] Dafou D, Grun B, Sinclair J, Lawrenson K, Benjamin EC, Hogdall E, Kruger-Kjaer S, Christensen L, Sowter HM, Al-Attar A, et al. (2010). Microcell-mediated chromosome transfer identifies EPB41L3 as a functional suppressor of epithelial ovarian cancers. *Neoplasia* **12**, 579–589.

[16] Defresne F, Bouzin C, Guilbaud C, Dieu M, Delaive E, Michiels C, Raes M, and Feron O (2010). Differential influence of anticancer treatments and angiogenesis on the seric titer of autoantibody used as tumor and metastasis biomarker. *Neoplasia* **12**, 562–570.

[17] Elo TD, Valve EM, Seppanen JA, Vuorikoski HJ, Makela SI, Poutanen M, Kujala PM, and Harkonen PL (2010). Stromal activation associated with development of prostate cancer in prostate-targeted fibroblast growth factor 8b transgenic mice. *Neoplasia* **12**, 915–927.

[18] Ericsson AC, Myles M, Davis W, Ma L, Lewis M, Maggio-Price L, and Franklin C (2010). Noninvasive detection of inflammation-associated colon cancer in a mouse model. *Neoplasia* **12**, 1054–1065.

[19] Fan S, Li Y, Yue P, Khuri FR, and Sun SY (2010). The eIF4E/eIF4G interaction inhibitor 4EGI-1 augments TRAIL-mediated apoptosis through c-FLIP down-regulation and DR5 induction independent of inhibition of cap-dependent protein translation. *Neoplasia* **12**, 346–356.

[20] Fournier PG, Stresing V, Ebetino FH, and Clezardin P (2010). How do bisphosphonates inhibit bone metastasis *in vivo*? *Neoplasia* **12**, 571–578.

[21] Fujita H, Ohuchida K, Mizumoto K, Itaba S, Ito T, Nakata K, Yu J, Kayashima T, Souzaki R, Tajiri T, et al. (2010). Gene expression levels as predictive markers of outcome in pancreatic cancer after gemcitabine-based adjuvant chemotherapy. *Neoplasia* **12**, 807–817.

[22] Fukunishi N, Katoh I, Tomimori Y, Tsukinoki K, Hata R, Nakao A, Ikawa Y, and Kurata S (2010). Induction of ΔNp63 by the newly identified keratinocyte-specific transforming growth factor β signaling pathway with Smad2 and IκB kinase α in squamous cell carcinoma. *Neoplasia* **12**, 969–979.

[23] Gadji M, Fortin D, Tsanaclis AM, Garini Y, Katzir N, Wienburg Y, Yan J, Klewes L, Klonisch T, Drouin R, et al. (2010). Three-dimensional nuclear telomere architecture is associated with differential time to progression and overall survival in glioblastoma patients. *Neoplasia* **12**, 183–191.

[24] Gandhirajan RK, Staib PA, Minke K, Gehrke I, Plickert G, Schlosser A, Schmitt EK, Hallek M, and Kreuzer KA (2010). Small molecule inhibitors of Wnt/β-catenin/lef-1 signaling induces apoptosis in chronic lymphocytic leukemia cells *in vitro* and *in vivo*. *Neoplasia* **12**, 326–335.

[25] Gong H, Kovar J, Little G, Chen H, and Olive DM (2010). *In vivo* imaging of xenograft tumors using an epidermal growth factor receptor–specific affibody molecule labeled with a near-infrared fluorophore. *Neoplasia* **12**, 139–149.

[26] Grinberg-Rashi H, Cytron S, Gelman-Kohan Z, Litmanovitch T, and Avivi L (2010). Replication timing aberrations and aneuploidy in peripheral blood lymphocytes of breast cancer patients. *Neoplasia* **12**, 668–674.

[27] Hagmann W, Jesnowski R, and Lohr JM (2010). Interdependence of gemcitabine treatment, transporter expression, and resistance in human pancreatic carcinoma cells. *Neoplasia* **12**, 740–747.

[28] Hail N Jr, Chen P, and Bushman LR (2010). Teriflunomide (leflunomide) promotes cytostatic, antioxidant, and apoptotic effects in transformed prostate epithelial cells: evidence supporting a role for teriflunomide in prostate cancer chemoprevention. *Neoplasia* **12**, 464–475.

[29] Halin S, Rudolfsson SH, Doll JA, Crawford SE, Wikstrom P, and Bergh A (2010). Pigment epithelium–derived factor stimulates tumor macrophage recruitment and is downregulated by the prostate tumor microenvironment. *Neoplasia* **12**, 336–345.

[30] Hatanpaa KJ, Burma S, Zhao D, and Habib AA (2010). Epidermal growth factor receptor in glioma: signal transduction, neuropathology, imaging, and radioresistance. *Neoplasia* **12**, 675–684.

[31] Hawcroft G, Loadman PM, Belluzzi A, and Hull MA (2010). Effect of eicosapentaenoic acid on E-type prostaglandin synthesis and EP4 receptor signaling in human colorectal cancer cells. *Neoplasia* **12**, 618–627.

[32] Hayashi Y, Molina JR, Hamilton SR, and Georgescu MM (2010). NHERF1/EBP50 is a new marker in colorectal cancer. *Neoplasia* **12**, 1013–1022.

[33] Hembruff SL, Jokar I, Yang L, and Cheng N (2010). Loss of transforming growth factor-β signaling in mammary fibroblasts enhances CCL2 secretion to promote mammary tumor progression through macrophage-dependent and -independent mechanisms. *Neoplasia* **12**, 425–433.

[34] Hoffmann AC, Wild P, Leicht C, Bertz S, Danenberg KD, Danenberg PV, Stohr R, Stockle M, Lehmann J, Schuler M, et al. (2010). MDR1 and ERCC1 expression predict outcome of patients with locally advanced bladder cancer receiving adjuvant chemotherapy. *Neoplasia* **12**, 628–636.

[35] Hotz B, Backer MV, Backer JM, Buhr HJ, and Hotz HG (2010). Specific targeting of tumor endothelial cells by a shiga-like toxin-vascular endothelial growth factor fusion protein as a novel treatment strategy for pancreatic cancer. *Neoplasia* **12**, 797–806.

[36] Hu Y, Sun H, Owens RT, Gu Z, Wu J, Chen YQ, O'Flaherty JT, and Edwards IJ (2010). Syndecan-1–dependent suppression of PDK1/Akt/bad signaling by docosahexaenoic acid induces apoptosis in prostate cancer. *Neoplasia* **12**, 826–836.

[37] Kadara H, Fujimoto J, Men T, Ye X, Lotan D, Lee JS, and Lotan R (2010). A Gprc5a tumor suppressor loss of expression signature is conserved, prevalent, and associated with survival in human lung adenocarcinomas. *Neoplasia* **12**, 499–505.

[38] Kahali S, Sarcar B, Fang B, Williams ES, Koomen JM, Tofilon PJ, and Chinnaiyan P (2010). Activation of the unfolded protein response contributes toward the antitumor activity of vorinostat. *Neoplasia* **12**, 80–86.

[39] Kakkad SM, Solaiyappan M, O'Rourke B, Stasinopoulos I, Ackerstaff E, Raman V, Bhujwalla ZM, and Glunde K (2010). Hypoxic tumor microenvironments reduce collagen I fiber density. *Neoplasia* **12**, 608–617.

[40] Kannan N, Kang J, Kong X, Tang J, Perry JK, Mohankumar KM, Miller LD, Liu ET, Mertani HC, Zhu T, et al. (2010). Trefoil factor 3 is oncogenic and mediates anti-estrogen resistance in human mammary carcinoma. *Neoplasia* **12**, 1041–1053.

[41] Kato T, Ueda Y, Kinoh H, Yoneyama Y, Matsunaga A, Komaru A, Harada Y, Suzuki H, Komiya A, Shibata S, et al. (2010). RIG-I helicase–independent pathway in sendai virus–activated dendritic cells is critical for preventing lung metastasis of AT6.3 prostate cancer. *Neoplasia* **12**, 906–914.

[42] Kaur M, Velmurugan B, Tyagi A, Agarwal C, Singh RP, and Agarwal R (2010). Silibinin suppresses growth of human colorectal carcinoma SW480 cells in culture and xenograft through down-regulation of β-catenin–dependent signaling. *Neoplasia* **12**, 415–424.

[43] Kerr BA, Miocinovic R, Smith AK, Klein EA, and Byzova TV (2010). Comparison of tumor and microenvironment secretomes in plasma and in platelets during prostate cancer growth in a xenograft model. *Neoplasia* **12**, 388–396.

[44] Klover PJ, Muller WJ, Robinson GW, Pfeiffer RM, Yamaji D, and Hennighausen L (2010). Loss of STAT1 from mouse mammary epithelium results in an increased Neu-induced tumor burden. *Neoplasia* **12**, 899–905.

[45] Lafferty-Whyte K, Bilsland A, Hoare SF, Burns S, Zaffaroni N, Cairney CJ, and Keith WN (2010). TCEAL7 inhibition of c-Myc activity in alternative lengthening of telomeres regulates hTERT expression. *Neoplasia* **12**, 405–414.

[46] Lamoral-Theys D, Le Mercier M, Le Calve B, Rynkowski MA, Bruyere C, Decaestecker C, Haibe-Kains B, Bontempi G, Dubois J, Lefranc F, et al. (2010). Long-term temozolomide treatment induces marked amino metabolism modifications and an increase in TMZ sensitivity in Hs683 oligodendroglioma cells. *Neoplasia* **12**, 69–79.

[47] Langenberg MH, Witteveen PO, Lankheet NA, Roodhart JM, Rosing H, van den Heuvel IJ, Beijnen JH, and Voest EE (2010). Phase 1 study of combination treatment with PTK 787/ZK 222584 and cetuximab for patients with advanced solid tumors: safety, pharmacokinetics, pharmacodynamics analysis. *Neoplasia* **12**, 206–213.

[48] Lawrenson K, Grun B, Benjamin E, Jacobs IJ, Dafou D, and Gayther SA (2010). Senescent fibroblasts promote neoplastic transformation of partially transformed ovarian epithelial cells in a three-dimensional model of early stage ovarian cancer. *Neoplasia* **12**, 317–325.

[49] Le Calve B, Rynkowski M, Le Mercier M, Bruyere C, Lonez C, Gras T, Haibe-Kains B, Bontempi G, Decaestecker C, Ruysschaert JM, et al. (2010). Long-term *in vitro* treatment of human glioblastoma cells with temozolomide increases resistance *in vivo* through up-regulation of GLUT transporter and aldo-keto reductase enzyme AKR1C expression. *Neoplasia* **12**, 727–739.

[50] Lee PC, Kakadiya R, Su TL, and Lee TC (2010). Combination of bifunctional alkylating agent and arsenic trioxide synergistically suppresses the growth of drug-resistant tumor cells. *Neoplasia* **12**, 376–387.

[51] Li CH, Cheng YW, Liao PL, and Kang JJ (2010). Translocation of p53 to mitochondria is regulated by its lipid binding property to anionic phospholipids and it participates in cell death control. *Neoplasia* **12**, 150–160.

[52] Li J, Favata M, Kelley JA, Caulder E, Thomas B, Wen X, Sparks RB, Arvanitis A, Rogers JD, Combs AP, et al. (2010). INCB16562, a JAK1/2 selective inhibitor, is efficacious against multiple myeloma cells and reverses the protective effects of cytokine and stromal cell support. *Neoplasia* **12**, 28–38.

[53] Li Y, Yue P, Deng X, Ueda T, Fukunaga R, Khuri FR, and Sun SY (2010). Protein phosphatase 2A negatively regulates eukaryotic initiation factor 4E phosphorylation and eIF4F assembly through direct dephosphorylation of Mnk and eIF4E. *Neoplasia* **12**, 848–855.

[54] Lin L, Hutzen B, Li PK, Ball S, Zuo M, DeAngelis S, Foust E, Sobo M, Friedman L, Bhasin D, et al. (2010). A novel small molecule, LLL12, inhibits STAT3 phosphorylation and activities and exhibits potent growth-suppressive activity in human cancer cells. *Neoplasia* **12**, 39–50.

[55] Lin Q, Balasubramanian K, Fan D, Kim SJ, Guo L, Wang H, Bar-Eli M, Aldape KD, and Fidler IJ (2010). Reactive astrocytes protect melanoma cells from chemotherapy by sequestering intracellular calcium through gap junction communication channels. *Neoplasia* **12**, 748–754.

[56] Liu Y, Laszlo C, Liu W, Chen X, Evans SC, and Wu S (2010). Regulation of G_1 arrest and apoptosis in hypoxia by PERK and GCN2-mediated eIF2α phosphorylation. *Neoplasia* **12**, 61–68.

[57] Lyu MA, Rai D, Ahn KS, Sung B, Cheung LH, Marks JW, Aggarwal BB, Aguiar RC, Gandhi V, and Rosenblum MG (2010). The rGel/BLyS fusion toxin inhibits diffuse large B-cell lymphoma growth *in vitro* and *in vivo*. *Neoplasia* **12**, 366–375.

[58] Maret D, Gruzglin E, Sadr MS, Siu V, Shan W, Koch AW, Seidah NG, Del Maestro RF, and Colman DR (2010). Surface expression of precursor N-cadherin promotes tumor cell invasion. *Neoplasia* **12**, 1066–1080.

[59] McClaine RJ, Marshall AM, Wagh PK, and Waltz SE (2010). Ron receptor tyrosine kinase activation confers resistance to tamoxifen in breast cancer cell lines. *Neoplasia* **12**, 650–658.

[60] Meister S, Frey B, Lang VR, Gaipl US, Schett G, Schlotzer-Schrehardt U, and Voll RE (2010). Calcium channel blocker verapamil enhances endoplasmic reticulum stress and cell death induced by proteasome inhibition in myeloma cells. *Neoplasia* **12**, 550–561.

[61] Molina JR, Hayashi Y, Stephens C, and Georgescu MM (2010). Invasive glioblastoma cells acquire stemness and increased Akt activation. *Neoplasia* **12**, 453–463.

[62] Mongiat M, Marastoni S, Ligresti G, Lorenzon E, Schiappacassi M, Perris R, Frustaci S, and Colombatti A (2010). The extracellular matrix glycoprotein elastin microfibril interface located protein 2: a dual role in the tumor microenvironment. *Neoplasia* **12**, 294–304.

[63] Morrissey C, Brown LG, Pitts TE, Vessella RL, and Corey E (2010). Bone morphogenetic protein 7 is expressed in prostate cancer metastases and its effects on prostate tumor cells depend on cell phenotype and the tumor microenvironment. *Neoplasia* **12**, 192–205.

[64] Nakahara Y, Northcott PA, Li M, Kongkham PN, Smith C, Yan H, Croul S, Ra YS, Eberhart C, Huang A, et al. (2010). Genetic and epigenetic inactivation of Kruppel-like factor 4 in medulloblastoma. *Neoplasia* **12**, 20–27.

[65] Paone A, Galli R, Gabellini C, Lukashev D, Starace D, Gorlach A, De Cesaris P, Ziparo E, Del Bufalo D, Sitkovsky MV, et al. (2010). Toll-like receptor 3 regulates angiogenesis and apoptosis in prostate cancer cell lines through hypoxia-inducible factor 1α. *Neoplasia* **12**, 539–549.

[66] Park K, Tomlins SA, Mudaliar KM, Chiu YL, Esgueva R, Mehra R, Suleman K, Varambally S, Brenner JC, MacDonald T, et al. (2010). Antibody-based detection of ERG rearrangement-positive prostate cancer. *Neoplasia* **12**, 590–598.

[67] Press JZ, Wurz K, Norquist BM, Lee MK, Pennil C, Garcia R, Welcsh P, Goff BA, and Swisher EM (2010). Identification of a preneoplastic gene expression profile in tubal epithelium of BRCA1 mutation carriers. *Neoplasia* **12**, 993–1002.

[68] Rabbani SA, Ateeq B, Arakelian A, Valentino ML, Shaw DE, Dauffenbach LM, Kerfoot CA, and Mazar AP (2010). An anti-urokinase plasminogen activator receptor antibody (ATN-658) blocks prostate cancer invasion, migration, growth, and experimental skeletal metastasis *in vitro* and *in vivo*. *Neoplasia* **12**, 778–788.

[69] Rantala JK, Edgren H, Lehtinen L, Wolf M, Kleivi K, Vollan HK, Aaltola AR, Laasola P, Kilpinen S, Saviranta P, et al. (2010). Integrative functional genomics analysis of sustained polyploidy phenotypes in breast cancer cells identifies an oncogenic profile for GINS2. *Neoplasia* **12**, 877–888.

[70] Rao G, Liu D, Xing M, Tauler J, Prinz RA, and Xu X (2010). Induction of heparanase-1 expression by mutant B-Raf kinase: role of GA binding protein in heparanase-1 promoter activation. *Neoplasia* **12**, 946–956.

[71] Rebhun RB, Cheng H, Gershenwald JE, Fan D, Fidler IJ, and Langley RR (2010). Constitutive expression of the α4 integrin correlates with tumorigenicity and lymph node metastasis of the B16 murine melanoma. *Neoplasia* **12**, 173–182.

[72] Rehemtulla A (2010). Dinosaurs and ancient civilizations: reflections on the treatment of cancer. *Neoplasia* **12**, 957–968.

[73] Rickman DS, Chen YB, Banerjee S, Pan Y, Yu J, Vuong T, Perner S, Lafargue CJ, Mertz KD, Setlur SR, et al. (2010). ERG cooperates with androgen receptor in regulating trefoil factor 3 in prostate cancer disease progression. *Neoplasia* **12**, 1031–1040.

[74] Ripka S, Riedel J, Neesse A, Griesmann H, Buchholz M, Ellenrieder V, Moeller F, Barth P, Gress TM, and Michl P (2010). Glutamate receptor GRIA3—target of CUX1 and mediator of tumor progression in pancreatic cancer. *Neoplasia* **12**, 659–667.

[75] Roe K, Seierstad T, Kristian A, Mikalsen LT, Maelandsmo GM, van der Kogel AJ, Ree AH, and Olsen DR (2010). Longitudinal magnetic resonance imaging-based assessment of vascular changes and radiation response in androgen-sensitive prostate carcinoma xenografts under androgen-exposed and androgen-deprived conditions. *Neoplasia* **12**, 818–825.

[76] Rofstad EK and Mathiesen B (2010). Metastasis in melanoma xenografts is associated with tumor microvascular density rather than extent of hypoxia. *Neoplasia* **12**, 889–898.

[77] Roodhart JM, Langenberg MH, Vermaat JS, Lolkema MP, Baars A, Giles RH, Witteveen EO, and Voest EE (2010). Late release of circulating endothelial cells and endothelial progenitor cells after chemotherapy predicts response and survival in cancer patients. *Neoplasia* **12**, 87–94.

[78] Rothweiler F, Michaelis M, Brauer P, Otte J, Weber K, Fehse B, Doerr HW, Wiese M, Kreuter J, Al-Abed Y, et al. (2010). Anticancer effects of the nitric oxide–modified saquinavir derivative saquinavir-NO against multidrug-resistant cancer cells. *Neoplasia* **12**, 1023–1030.

[79] Schneider M, Wortmann M, Mandal PK, Arpornchayanon W, Jannasch K, Alves F, Strieth S, Conrad M, and Beck H (2010). Absence of glutathione peroxidase 4 affects tumor angiogenesis through increased 12/15-lipoxygenase activity. *Neoplasia* **12**, 254–263.

[80] Sengupta S, Nandi S, Hindi ES, Wainwright DA, Han Y, and Lesniak MS (2010). Short hairpin RNA–mediated fibronectin knockdown delays tumor growth in a mouse glioma model. *Neoplasia* **12**, 837–847.

[81] Shiozawa Y, Pedersen EA, Patel LR, Ziegler AM, Havens AM, Jung Y, Wang J, Zalucha S, Loberg RD, Pienta KJ, et al. (2010). GAS6/AXL axis regulates prostate cancer invasion, proliferation, and survival in the bone marrow niche. *Neoplasia* **12**, 116–127.

[82] Silva AG, Graves HA, Guffei A, Ricca TI, Mortara RA, Jasiulionis MG, and Mai S (2010). Telomere-centromere–driven genomic instability contributes to karyotype evolution in a mouse model of melanoma. *Neoplasia* **12**, 11–19.

[83] St Germain C, Niknejad N, Ma L, Garbuio K, Hai T, and Dimitroulakos J (2010). Cisplatin induces cytotoxicity through the mitogen-activated protein kinase pathways and activating transcription factor 3. *Neoplasia* **12**, 527–538.

[84] Tai JH, Tessier J, Ryan AJ, Hoffman L, Chen X, and Lee TY (2010). Assessment of acute antivascular effects of vandetanib with high-resolution dynamic contrast-enhanced computed tomographic imaging in a human colon tumor xenograft model in the nude rat. *Neoplasia* **12**, 697–707.

[85] Tang MK, Zhou HY, Yam JW, and Wong AS (2010). c-Met overexpression contributes to the acquired apoptotic resistance of nonadherent ovarian cancer cells through a cross talk mediated by phosphatidylinositol 3-kinase and extracellular signal–regulated kinase 1/2. *Neoplasia* **12**, 128–138.

[86] Tang TC, Man S, Lee CR, Xu P, and Kerbel RS (2010). Impact of metronomic UFT/cyclophosphamide chemotherapy and antiangiogenic drug assessed in a new preclinical model of locally advanced orthotopic hepatocellular carcinoma. *Neoplasia* **12**, 264–274.

[87] Tang TC, Man S, Xu P, Francia G, Hashimoto K, Emmenegger U, and Kerbel RS (2010). Development of a resistance-like phenotype to sorafenib by human hepatocellular carcinoma cells is reversible and can be delayed by metronomic UFT chemotherapy. *Neoplasia* **12**, 928–940.

[88] Tap WD, Gong KW, Dering J, Tseng Y, Ginther C, Pauletti G, Glaspy JA, Essner R, Bollag G, Hirth P, et al. (2010). Pharmacodynamic characterization of the efficacy signals due to selective BRAF inhibition with PLX4032 in malignant melanoma. *Neoplasia* **12**, 637–649.

[89] Tennis MA, Van Scoyk M, Heasley LE, Vandervest K, Weiser-Evans M, Freeman S, Keith RL, Simpson P, Nemenoff RA, and Winn RA (2010). Prostacyclin inhibits non–small cell lung cancer growth by a frizzled 9–dependent pathway that is blocked by secreted frizzled-related protein 1. *Neoplasia* **12**, 244–253.

[90] Tetsu O, Phuchareon J, Chou A, Cox DP, Eisele DW, and Jordan RC (2010). Mutations in the c-Kit gene disrupt mitogen-activated protein kinase signaling during tumor development in adenoid cystic carcinoma of the salivary glands. *Neoplasia* **12**, 708–717.

[91] Toneff MJ, Du Z, Dong J, Huang J, Sinai P, Forman J, Hilsenbeck S, Schiff R, Huang S, and Li Y (2010). Somatic expression of PyMT or activated ErbB2 induces estrogen-independent mammary tumorigenesis. *Neoplasia* **12**, 718–726.

[92] Tonks ID, Mould AW, Schroder WA, Cotterill A, Hayward NK, Walker GJ, and Kay GF (2010). Dual loss of rb1 and Trp53 in the adrenal medulla leads to spontaneous pheochromocytoma. *Neoplasia* **12**, 235–243.

[93] Tsunoda T, Takashima Y, Fujimoto T, Koyanagi M, Yoshida Y, Doi K, Tanaka Y, Kuroki M, Sasazuki T, and Shirasawa S (2010). Three-dimensionally specific inhibition of DNA repair–related genes by activated KRAS in colon crypt model. *Neoplasia* **12**, 397–404.

[94] Tysnes BB (2010). Tumor-initiating and -propagating cells: cells that we would like to identify and control. *Neoplasia* **12**, 506–515.

[95] Tzuman YC, Sapoznik S, Granot D, Nevo N, and Neeman M (2010). Peritoneal adhesion and angiogenesis in ovarian carcinoma are inversely regulated by hyaluronan: the role of gonadotropins. *Neoplasia* **12**, 51–60.

[96] van Houdt WJ, Hoogwater FJ, de Bruijn MT, Emmink BL, Nijkamp MW, Raats DA, van der Groep P, van Diest P, Borel Rinkes IH, and Kranenburg O (2010). Oncogenic KRAS desensitizes colorectal tumor cells to epidermal growth factor receptor inhibition and activation. *Neoplasia* **12**, 443–452.

[97] Velmurugan B, Singh RP, Kaul N, Agarwal R, and Agarwal C (2010). Dietary feeding of grape seed extract prevents intestinal tumorigenesis in $APC^{min/+}$ mice. *Neoplasia* **12**, 95–102.

[98] Wakasaki T, Masuda M, Niiro H, Jabbarzadeh-Tabrizi S, Noda K, Taniyama T, Komune S, and Akashi K (2010). A critical role of c-Cbl–interacting protein of 85 kDa in the development and progression of head and neck squamous cell carcinomas through the ras-ERK pathway. *Neoplasia* **12**, 789–796.

[99] Wang Q, Zhang W, Liu Q, Zhang X, Lv N, and Ye L (2010). A mutant of hepatitis B virus X protein (HBxΔ127) promotes cell growth through a positive feedback loop involving 5-lipoxygenase and fatty acid synthase. *Neoplasia* **12**, 103–115.

[100] Wang S, Huang J, He J, Wang A, Xu S, Huang SF, and Xiao S (2010). RPL41, a small ribosomal peptide deregulated in tumors, is essential for mitosis and centrosome integrity. *Neoplasia* **12**, 284–293.

[101] Watson RL, Spalding AC, Zielske SP, Morgan M, Kim AC, Bommer GT, Eldar-Finkelman H, Giordano T, Fearon ER, Hammer GD, et al. (2010). GSK3β and β-catenin modulate radiation cytotoxicity in pancreatic cancer. *Neoplasia* **12**, 357–365.

[102] Witteveen PO, van der Mijn KJ, Los M, Kronemeijer RH, Groenewegen G, and Voest EE (2010). Phase 1/2 study of atrasentan combined with pegylated liposomal doxorubicin in platinum-resistant recurrent ovarian cancer. *Neoplasia* **12**, 941–945.

[103] Wu Z, Owens C, Chandra N, Popovic K, Conaway M, and Theodorescu D (2010). RalBP1 is necessary for metastasis of human cancer cell lines. *Neoplasia* **12**, 1003–1012.

[104] Zhao H, Ou-Yang F, Chen IF, Hou MF, Yuan SS, Chang HL, Lee YC, Plattner R, Waltz SE, Ho SM, et al. (2010). Enhanced resistance to tamoxifen by the c-ABL proto-oncogene in breast cancer. *Neoplasia* **12**, 214–223.

[105] Zhou H, Liu Y, Cheung LH, Kim S, Zhang W, Mohamedali KA, Anand P, Hittelman WN, Aggarwal BB, and Rosenblum MG (2010). Characterization and mechanistic studies of a novel melanoma-targeting construct containing IκBa for specific inhibition of nuclear factor-κB activity. *Neoplasia* **12**, 766–777.

[106] Zillhardt M, Christensen JG, and Lengyel E (2010). An orally available small-molecule inhibitor of c-Met, PF-2341066, reduces tumor burden and metastasis in a preclinical model of ovarian cancer metastasis. *Neoplasia* **12**, 1–10.

[107] Agudelo-Garcia PA, De Jesus JK, Williams SP, Nowicki MO, Chiocca EA, Liyanarachchi S, Li PK, Lannutti JJ, Johnson JK, Lawler SE, et al. (2011). Glioma cell migration on three-dimensional nanofiber scaffolds is regulated by substrate topography and abolished by inhibition of STAT3 signaling. *Neoplasia* **13**, 831–840.

[108] Bekes EM, Deryugina EI, Kupriyanova TA, Zajac E, Botkjaer KA, Andreasen PA, and Quigley JP (2011). Activation of pro-uPA is critical for initial escape from the primary tumor and hematogenous dissemination of human carcinoma cells. *Neoplasia* **13**, 806–821.

[109] Berdiel-Acer M, Bohem ME, Lopez-Doriga A, Vidal A, Salazar R, Martinez-Iniesta M, Santos C, Sanjuan X, Villanueva A, and Mollevi DG (2011). Hepatic carcinoma–associated fibroblasts promote an adaptative response in colorectal cancer cells that inhibit proliferation and apoptosis: nonresistant cells die by nonapoptotic cell death. *Neoplasia* **13**, 931–946.

[110] Birbach A, Eisenbarth D, Kozakowski N, Ladenhauf E, Schmidt-Supprian M, and Schmid JA (2011). Persistent inflammation leads to proliferative neoplasia and loss of smooth muscle cells in a prostate tumor model. *Neoplasia* **13**, 692–703.

[111] Blando JM, Carbajal S, Abel E, Beltran L, Conti C, Fischer S, and DiGiovanni J (2011). Cooperation between Stat3 and Akt signaling leads to prostate tumor development in transgenic mice. *Neoplasia* **13**, 254–265.

[112] Borsig L, Vlodavsky I, Ishai-Michaeli R, Torri G, and Vismara E (2011). Sulfated hexasaccharides attenuate metastasis by inhibition of P-selectin and heparanase. *Neoplasia* **13**, 445–452.

[113] Candolfi M, Curtin JF, Yagiz K, Assi H, Wibowo MK, Alzadeh GE, Foulad D, Muhammad AK, Salehi S, Keech N, et al. (2011). B cells are critical to T-cell–mediated antitumor immunity induced by a combined immune-stimulatory/conditionally cytotoxic therapy for glioblastoma. *Neoplasia* **13**, 947–960.

[114] Canu B, Fioravanti A, Orlandi P, Di Desidero T, Ali G, Fontanini G, Di Paolo A, Del Tacca M, Danesi R, and Bocci G (2011). Irinotecan synergistically enhances the antiproliferative and proapoptotic effects of axitinib *in vitro* and improves its anticancer activity *in vivo*. *Neoplasia* **13**, 217–229.

[115] Ceteci F, Xu J, Ceteci S, Zanucco E, Thakur C, and Rapp UR (2011). Conditional expression of oncogenic C-RAF in mouse pulmonary epithelial cells reveals differential tumorigenesis and induction of autophagy leading to tumor regression. *Neoplasia* **13**, 1005–1018.

[116] Chia KM, Liu J, Francis GD, and Naderi A (2011). A feedback loop between androgen receptor and ERK signaling in estrogen receptor–negative breast cancer. *Neoplasia* **13**, 154–166.

[117] Cho HS, Kelly JD, Hayami S, Toyokawa G, Takawa M, Yoshimatsu M, Tsunoda T, Field HI, Neal DE, Ponder BA, et al. (2011). Enhanced expression of EHMT2 is involved in the proliferation of cancer cells through negative regulation of SIAH1. *Neoplasia* **13**, 676–684.

[118] Clapper ML, Hensley HH, Chang WC, Devarajan K, Nguyen MT, and Cooper HS (2011). Detection of colorectal adenomas using a bioactivatable probe specific for matrix metalloproteinase activity. *Neoplasia* **13**, 685–691.

[119] Colen CB, Shen Y, Ghoddoussi F, Yu P, Francis TB, Koch BJ, Monterey MD, Galloway MP, Sloan AE, and Mathupala SP (2011). Metabolic targeting of lactate efflux by malignant glioma inhibits invasiveness and induces necrosis: an *in vivo* study. *Neoplasia* **13**, 620–632.

[120] Conradt L, Godl K, Schaab C, Tebbe A, Eser S, Diersch S, Michalski CW, Kleeff J, Schnieke A, Schmid RM, et al. (2011). Disclosure of erlotinib as a multikinase inhibitor in pancreatic ductal adenocarcinoma. *Neoplasia* **13**, 1026–1034.

[121] Coulon A, Flahaut M, Muhlethaler-Mottet A, Meier R, Liberman J, Balmas-Bourloud K, Nardou K, Yan P, Tercier S, Joseph JM, et al. (2011). Functional sphere profiling reveals the complexity of neuroblastoma tumor-initiating cell model. *Neoplasia* **13**, 991–1004.

[122] Davison Z, de Blacquiere GE, Westley BR, and May FE (2011). Insulin-like growth factor–dependent proliferation and survival of triple-negative breast cancer cells: implications for therapy. *Neoplasia* **13**, 504–515.

[123] De Smaele E, Di Marcotullio L, Moretti M, Pelloni M, Occhione MA, Infante P, Cucchi D, Greco A, Pietrosanti L, Todorovic J, et al. (2011). Identification and characterization of KCASH2 and KCASH3, 2 novel Cullin3 adaptors suppressing histone deacetylase and Hedgehog activity in medulloblastoma. *Neoplasia* **13**, 374–385.

[124] De Vitis S, Sonia Treglia A, Ulianich L, Turco S, Terrazzano G, Lombardi A, Miele C, Garbi C, Beguinot F, and Di Jeso B (2011). Tyr phosphatase–mediated P-ERK inhibition suppresses senescence in EIA + v-*raf* transformed cells, which, paradoxically, are apoptosis-protected in a MEK-dependent manner. *Neoplasia* **13**, 120–130.

[125] Dean EJ, Cummings J, Roulston A, Berger M, Ranson M, Blackhall F, and Dive C (2011). Optimization of circulating biomarkers of obatoclax-induced cell death in patients with small cell lung cancer. *Neoplasia* **13**, 339–347.

[126] Doucette T, Rao G, Yang Y, Gumin J, Shinojima N, Bekele BN, Qiao W, Zhang W, and Lang FF (2011). Mesenchymal stem cells display tumor-specific tropism in an RCAS/Ntv-a glioma model. *Neoplasia* **13**, 716–725.

[127] Duignan IJ, Corcoran E, Pennello A, Plym MJ, Amatulli M, Claros N, Iacolina M, Youssoufian H, Witte L, Samakoglu S, et al. (2011). Pleiotropic stromal effects of vascular endothelial growth factor receptor 2 antibody therapy in renal cell carcinoma models. *Neoplasia* **13**, 49–59.

[128] Edrei Y, Gross E, Corchia N, Tsarfaty G, Galun E, Pappo O, and Abramovitch R (2011). Vascular profile characterization of liver tumors by magnetic resonance imaging using hemodynamic response imaging in mice. *Neoplasia* **13**, 244–253.

[129] Emmenegger U, Francia G, Chow A, Shaked Y, Kouri A, Man S, and Kerbel RS (2011). Tumors that acquire resistance to low-dose metronomic cyclophosphamide retain sensitivity to maximum tolerated dose cyclophosphamide. *Neoplasia* **13**, 40–48.

[130] Fendrich V, Oh E, Bang S, Karikari C, Ottenhof N, Bisht S, Lauth M, Brossart P, Katsanis N, Maitra A, et al. (2011). Ectopic overexpression of Sonic Hedgehog (Shh) induces stromal expansion and metaplasia in the adult murine pancreas. *Neoplasia* **13**, 923–930.

[131] Fendrich V, Schneider R, Maitra A, Jacobsen ID, Opfermann T, and Bartsch DK (2011). Detection of precursor lesions of pancreatic adenocarcinoma in PET-CT in a genetically engineered mouse model of pancreatic cancer. *Neoplasia* **13**, 180–186.

[132] Feng L, Sun X, Csizmadia E, Han L, Bian S, Murakami T, Wang X, Robson SC, and Wu Y (2011). Vascular CD39/ENTPD1 directly promotes tumor cell growth by scavenging extracellular adenosine triphosphate. *Neoplasia* **13**, 206–216.

[133] Gatza CE, Holtzhausen A, Kirkbride KC, Morton A, Gatza ML, Datto MB, and Blobe GC (2011). Type III TGF-β receptor enhances colon cancer cell migration and anchorage-independent growth. *Neoplasia* **13**, 758–770.

[134] Grohar PJ, Griffin LB, Yeung C, Chen QR, Pommier Y, Khanna C, Khan J, and Helman LJ (2011). Ecteinascidin 743 interferes with the activity of EWS-FLI1 in Ewing sarcoma cells. *Neoplasia* **13**, 145–153.

[135] Haim K, Weitzenfeld P, Meshel T, and Ben-Baruch A (2011). Epidermal growth factor and estrogen act by independent pathways to additively promote the release of the angiogenic chemokine CXCL8 by breast tumor cells. *Neoplasia* **13**, 230–243.

[136] Halder SK, Cho YJ, Datta A, Anumanthan G, Ham AJ, Carbone DP, and Datta PK (2011). Elucidating the mechanism of regulation of transforming growth factor β type II receptor expression in human lung cancer cell lines. *Neoplasia* **13**, 912–922.

[137] He Y, Cui Y, Wang W, Gu J, Guo S, Ma K, and Luo X (2011). Hypomethylation of the hsa-miR-191 locus causes high expression of hsa-mir-191 and promotes the epithelial-to-mesenchymal transition in hepatocellular carcinoma. *Neoplasia* **13**, 841–853.

[138] Iwamoto H, Torimura T, Nakamura T, Hashimoto O, Inoue K, Kurogi J, Niizeki T, Kuwahara R, Abe M, Koga H, et al. (2011). Metronomic S-1 chemotherapy and vandetanib: an efficacious and nontoxic treatment for hepatocellular carcinoma. *Neoplasia* **13**, 187–197.

[139] Jazaeri AA, Bryant JL, Park H, Li H, Dahiya N, Stoler MH, Ferriss JS, and Dutta A (2011). Molecular requirements for transformation of fallopian tube epithelial cells into serous carcinoma. *Neoplasia* **13**, 899–911.

[140] Jia L, Li H, and Sun Y (2011). Induction of p21-dependent senescence by an NAE inhibitor, MLN4924, as a mechanism of growth suppression. *Neoplasia* **13**, 561–569.

[141] Jiang Y, Boije M, Westermark B, and Uhrbom L (2011). PDGF-B can sustain self-renewal and tumorigenicity of experimental glioma-derived cancer-initiating cells by preventing oligodendrocyte differentiation. *Neoplasia* **13**, 492–503.

[142] Kang K, Oh SH, Yun JH, Jho EH, Kang JH, Batsuren D, Tunsag J, Park KH, Kim M, and Nho CW (2011). A novel topoisomerase inhibitor, daurinol, suppresses growth of HCT116 cells with low hematological toxicity compared to etoposide. *Neoplasia* **13**, 1043–1057.

[143] Kao HW, Sanada M, Liang DC, Lai CL, Lee EH, Kuo MC, Lin TL, Shih YS, Wu JH, Huang CF, et al. (2011). A high occurrence of acquisition and/or expansion of C-CBL mutant clones in the progression of high-risk myelodysplastic syndrome to acute myeloid leukemia. *Neoplasia* **13**, 1035–1042.

[144] Karabela S, Kairi CA, Magkouta S, Psallidas I, Moschos C, Stathopoulos I, Zakynthinos SG, Roussos C, Kalomenidis I, and Stathopoulos GT (2011). Neutralization of tumor necrosis factor bioactivity ameliorates urethane-induced pulmonary oncogenesis in mice. *Neoplasia* **13**, 1143–1151.

[145] Kashef K, Radhakrishnan R, Lee CM, Reddy EP, and Dhanasekaran DN (2011). Neoplastic transformation induced by the gep oncogenes involves the scaffold protein JNK-interacting leucine zipper protein. *Neoplasia* **13**, 358–364.

[146] Katoh I, Mirova A, Kurata S, Murakami Y, Horikawa K, Nakakuki N, Sakai T, Hashimoto K, Maruyama A, Yonaga T, et al. (2011). Activation of the long terminal repeat of human endogenous retrovirus K by melanoma-specific transcription factor MITF-M. *Neoplasia* **13**, 1081–1092.

[147] Kim SJ, Kim JS, Kim SW, Brantley E, Yun SJ, He J, Maya M, Zhang F, Wu Q, Lehembre F, et al. (2011). Macitentan (ACT-064992), a tissue-targeting endothelin receptor antagonist, enhances therapeutic efficacy of paclitaxel by modulating survival pathways in orthotopic models of metastatic human ovarian cancer. *Neoplasia* **13**, 167–179.

[148] Kim SJ, Kim JS, Park ES, Lee JS, Lin Q, Langley RR, Maya M, He J, Kim SW, Weihua Z, et al. (2011). Astrocytes upregulate survival genes in tumor cells and induce protection from chemotherapy. *Neoplasia* **13**, 286–298.

[149] Kirabo A, Park SO, Majumder A, Gali M, Reinhard MK, Wamsley HL, Zhao ZJ, Cogle CR, Bisht KS, Keseru GM, et al. (2011). The Jak2 inhibitor, G6, alleviates Jak2-V617F–mediated myeloproliferative neoplasia by providing significant therapeutic efficacy to the bone marrow. *Neoplasia* **13**, 1058–1068.

[150] Klose A, Waerzeggers Y, Monfared P, Vukicevic S, Kaijzel EL, Winkeler A, Wickenhauser C, Lowik CW, and Jacobs AH (2011). Imaging bone morphogenetic protein 7 induced cell cycle arrest in experimental gliomas. *Neoplasia* **13**, 276–285.

[151] Knobel PA, Kotov IN, Felley-Bosco E, Stahel RA, and Marti TM (2011). Inhibition of REV3 expression induces persistent DNA damage and growth arrest in cancer cells. *Neoplasia* **13**, 961–970.

[152] Kong J, Crissey MA, Stairs DB, Sepulveda AR, and Lynch JP (2011). Cox2 and β-catenin/T-cell factor signaling intestinalize human esophageal keratinocytes when cultured under organotypic conditions. *Neoplasia* **13**, 792–805.

[153] Kurhanewicz J, Vigneron DB, Brindle K, Chekmenev EY, Comment A, Cunningham CH, Deberardinis RJ, Green GG, Leach MO, Rajan SS, et al. (2011). Analysis of cancer metabolism by imaging hyperpolarized nuclei: prospects for translation to clinical research. *Neoplasia* **13**, 81–97.

[154] Ladhani O, Sanchez-Martinez C, Orgaz JL, Jimenez B, and Volpert OV (2011). Pigment epithelium–derived factor blocks tumor extravasation by suppressing amoeboid morphology and mesenchymal proteolysis. *Neoplasia* **13**, 633–642.

[155] Laukens B, Jennewein C, Schenk B, Vanlangenakker N, Schier A, Cristofanon S, Zobel K, Deshayes K, Vucic D, Jeremias I, et al. (2011). Smac mimetic bypasses apoptosis resistance in FADD- or caspase-8–deficient cells by priming for tumor necrosis factor α–induced necroptosis. *Neoplasia* **13**, 971–979.

[156] Laulajainen M, Muranen T, Nyman TA, Carpen O, and Gronholm M (2011). Multistep phosphorylation by oncogenic kinases enhances the degradation of the NF2 tumor suppressor merlin. *Neoplasia* **13**, 643–652.

[157] Li Y, Ye X, Liu J, Zha J, and Pei L (2011). Evaluation of EML4-ALK fusion proteins in non–small cell lung cancer using small molecule inhibitors. *Neoplasia* **13**, 1–11.

[158] Liu Y, Norton JT, Witschi MA, Xu Q, Lou G, Wang C, Appella DH, Chen Z, and Huang S (2011). Methoxyethylamino-numonafide is an efficacious and minimally toxic amonafide derivative in murine models of human cancer. *Neoplasia* **13**, 453–460.

[159] Lo Monaco E, Tremante E, Cerboni C, Melucci E, Sibilio L, Zingoni A, Nicotra MR, Natali PG, and Giacomini P (2011). Human leukocyte antigen E contributes to protect tumor cells from lysis by natural killer cells. *Neoplasia* **13**, 822–830.

[160] Lonigro RJ, Grasso CS, Robinson DR, Jing X, Wu YM, Cao X, Quist MJ, Tomlins SA, Pienta KJ, and Chinnaiyan AM (2011). Detection of somatic copy number alterations in cancer using targeted exome capture sequencing. *Neoplasia* **13**, 1019–1025.

[161] Lorch G, Viatchenko-Karpinski S, Ho HT, Dirksen WP, Toribio RE, Foley J, Gyorke S, and Rosol TJ (2011). The calcium-sensing receptor is necessary for the rapid development of hypercalcemia in human lung squamous cell carcinoma. *Neoplasia* **13**, 428–438.

[162] Lu H, Liu P, Pan Y, and Huang H (2011). Inhibition of cyclin-dependent kinase phosphorylation of FOXO1 and prostate cancer cell growth by a peptide derived from FOXO1. *Neoplasia* **13**, 854–863.

[163] Lu Z, Ercolano E, Ammoun S, Schmid MC, Barczyk M, and Hanemann CO (2011). Merlin-deficient human tumours show loss of contact inhibition, activation of Wnt/β-catenin signaling linked to the PDGFR/Src and Rac/PAK pathways. *Neoplasia* **13**, 1101–1112.

[164] Ly P, Eskiocak U, Kim SB, Roig AI, Hight SK, Lulla DR, Zou YS, Batten K, Wright WE, and Shay JW (2011). Characterization of aneuploid populations with trisomy 7 and 20 derived from diploid human colonic epithelial cells. *Neoplasia* **13**, 348–357.

[165] Madhavan S, Gusev Y, Harris M, Tanenbaum DM, Gauba R, Bhuvaneshwar K, Shinohara A, Rosso K, Carabet LA, Song L, et al. (2011). G-DOC: a systems medicine platform for personalized oncology. *Neoplasia* **13**, 771–783.

[166] Margolin DA, Silinsky J, Grimes C, Spencer N, Aycock M, Green H, Cordova J, Davis NK, Driscoll T, and Li L (2011). Lymph node stromal cells enhance drug-resistant colon cancer cell tumor formation through SDF-1α/CXCR4 paracrine signaling. *Neoplasia* **13**, 874–886.

[167] McCabe NP, Kerr BA, Madajka M, Vasanji A, and Byzova TV (2011). Augmented osteolysis in SPARC-deficient mice with bone-residing prostate cancer. *Neoplasia* **13**, 31–39.

[168] Meng F, Zhang H, Liu G, Kreike B, Chen W, Sethi S, Miller FR, and Wu G (2011). p38γ mitogen-activated protein kinase contributes to oncogenic properties maintenance and resistance to poly (ADP-ribose)-polymerase-1 inhibition in breast cancer. *Neoplasia* **13**, 472–482.

[169] Montano N, Cenci T, Martini M, D'Alessandris QG, Pelacchi F, Ricci-Vitiani L, Maira G, De Maria R, Larocca JM, and Pallini R (2011). Expression of EGFRvIII in glioblastoma: prognostic significance revisited. *Neoplasia* **13**, 1113–1121.

[170] Morello S, Sorrentino R, Montinaro A, Luciano A, Maiolino P, Ngkelo A, Arra C, Adcock IM, and Pinto A (2011). NK1.1$^+$ cells and CD8$^+$ T cells mediate the antitumor activity of Cl-IB-MECA in a mouse melanoma model. *Neoplasia* **13**, 365–373.

[171] Naderi EH, Jochemsen AG, Blomhoff HK, and Naderi S (2011). Activation of cAMP signaling interferes with stress-induced p53 accumulation in ALL-derived cells by promoting the interaction between p53 and HDM2. *Neoplasia* **13**, 653–663.

[172] Ou WB, Hubert C, Corson JM, Bueno R, Flynn DL, Sugarbaker DJ, and Fletcher JA (2011). Targeted inhibition of multiple receptor tyrosine kinases in mesothelioma. *Neoplasia* **13**, 12–22.

[173] Park JH, Katagiri T, Chung S, Kijima K, and Nakamura Y (2011). Polypeptide *N*-acetylgalactosaminyltransferase 6 disrupts mammary acinar morphogenesis through *O*-glycosylation of fibronectin. *Neoplasia* **13**, 320–326.

[174] Pernicova Z, Slabakova E, Kharaishvili G, Bouchal J, Kral M, Kunicka Z, Machala M, Kozubik A, and Soucek K (2011). Androgen depletion induces senescence in prostate cancer cells through down-regulation of Skp2. *Neoplasia* **13**, 526–536.

[175] Rattan R, Graham RP, Maguire JL, Giri S, and Shridhar V (2011). Metformin suppresses ovarian cancer growth and metastasis with enhancement of cisplatin cytotoxicity *in vivo*. *Neoplasia* **13**, 483–491.

[176] Ray D, Ahsan A, Helman A, Chen G, Hegde A, Gurjar SR, Zhao L, Kiyokawa H, Beer DG, Lawrence TS, et al. (2011). Regulation of EGFR protein stability by the HECT-type ubiquitin ligase SMURF2. *Neoplasia* **13**, 570–578.

[177] Ray D, Terao Y, Christov K, Kaldis P, and Kiyokawa H (2011). Cdk2-null mice are resistant to ErbB-2-induced mammary tumorigenesis. *Neoplasia* **13**, 439–444.

[178] Ray P, Lewin SA, Mihalko LA, Schmidt BT, Luker KE, and Luker GD (2011). Noninvasive imaging reveals inhibition of ovarian cancer by targeting CXCL12-CXCR4. *Neoplasia* **13**, 1152–1161.

[179] Ruddell A, Harrell MI, Furuya M, Kirschbaum SB, and Iritani BM (2011). B lymphocytes promote lymphogenous metastasis of lymphoma and melanoma. *Neoplasia* **13**, 748–757.

[180] Samassekou O, Li H, Hebert J, Ntwari A, Wang H, Cliche CG, Bouchard E, Huang S, and Yan J (2011). Chromosome arm–specific long telomeres: a new clonal event in primary chronic myelogenous leukemia cells. *Neoplasia* **13**, 550–560.

[181] Sampetrean O, Saga I, Nakanishi M, Sugihara E, Fukaya R, Onishi N, Osuka S, Akahata M, Kai K, Sugimoto H, et al. (2011). Invasion precedes tumor mass formation in a malignant brain tumor model of genetically modified neural stem cells. *Neoplasia* **13**, 784–791.

[182] Schauer IG, Sood AK, Mok S, and Liu J (2011). Cancer-associated fibroblasts and their putative role in potentiating the initiation and development of epithelial ovarian cancer. *Neoplasia* **13**, 393–405.

[183] Seth P, Grant A, Tang J, Vinogradov E, Wang X, Lenkinski R, and Sukhatme VP (2011). On-target inhibition of tumor fermentative glycolysis as visualized by hyperpolarized pyruvate. *Neoplasia* **13**, 60–71.

[184] Shahzad MM, Mangala LS, Han HD, Lu C, Bottsford-Miller J, Nishimura M, Mora EM, Lee JW, Stone RL, Pecot CV, et al. (2011). Targeted delivery of small interfering RNA using reconstituted high-density lipoprotein nanoparticles. *Neoplasia* **13**, 309–319.

[185] Shibata T, Kokubu A, Saito S, Narisawa-Saito M, Sasaki H, Aoyagi K, Yoshimatsu Y, Tachimori Y, Kushima R, Kiyono T, et al. (2011). NRF2 mutation confers malignant potential and resistance to chemoradiation therapy in advanced esophageal squamous cancer. *Neoplasia* **13**, 864–873.

[186] Smith SC, Havaleshko DM, Moon K, Baras AS, Lee J, Bekiranov S, Burke DJ, and Theodorescu D (2011). Use of yeast chemigenomics and COXEN informatics in preclinical evaluation of anticancer agents. *Neoplasia* **13**, 72–80.

[187] Soucek L, Buggy JJ, Kortlever R, Adimoolam S, Monclus HA, Allende MT, Swigart LB, and Evan GI (2011). Modeling pharmacological inhibition of mast cell degranulation as a therapy for insulinoma. *Neoplasia* **13**, 1093–1100.

[188] Stafford JH and Thorpe PE (2011). Increased exposure of phosphatidylethanolamine on the surface of tumor vascular endothelium. *Neoplasia* **13**, 299–308.

[189] Starlinger P, Brugger P, Reiter C, Schauer D, Sommerfeldt S, Tamandl D, Kuehrer I, Schoppmann SF, Gnant M, and Brostjan C (2011). Discrimination between circulating endothelial cells and blood cell populations with overlapping phenotype reveals distinct regulation and predictive potential in cancer therapy. *Neoplasia* **13**, 980–990.

[190] Starlinger P, Brugger P, Schauer D, Sommerfeldt S, Tamandl D, Kuehrer I, Schoppmann SF, Gnant M, and Brostjan C (2011). Myelosuppression of thrombocytes and monocytes is associated with a lack of synergy between chemotherapy and anti-VEGF treatment. *Neoplasia* **13**, 419–427.

[191] Stein U, Arlt F, Smith J, Sack U, Herrmann P, Walther W, Lemm M, Fichtner I, Shoemaker RH, and Schlag PM (2011). Intervening in β-catenin signaling by sulindac inhibits S100A4-dependent colon cancer metastasis. *Neoplasia* **13**, 131–144.

[192] Stronach EA, Chen M, Maginn EN, Agarwal R, Mills GB, Wasan H, and Gabra H (2011). DNA-PK mediates AKT activation and apoptosis inhibition in clinically acquired platinum resistance. *Neoplasia* **13**, 1069–1080.

[193] Subramanian C, Jarzembowski JA, Opipari AW Jr, Castle VP, and Kwok RP (2011). HDAC6 deacetylates Ku70 and regulates Ku70-Bax binding in neuroblastoma. *Neoplasia* **13**, 726–734.

[194] Sun CK, Chua MS, He J, and So SK (2011). Suppression of glypican 3 inhibits growth of hepatocellular carcinoma cells through up-regulation of TGF-β2. *Neoplasia* **13**, 735–747.

[195] Tang Y, Parmakhtiar B, Simoneau AR, Xie J, Fruehauf J, Lilly M, and Zi X (2011). Lycopene enhances docetaxel's effect in castration-resistant prostate cancer associated with insulin-like growth factor I receptor levels. *Neoplasia* **13**, 108–119.

[196] Taylor MA, Amin JD, Kirschmann DA, and Schiemann WP (2011). Lysyl oxidase contributes to mechanotransduction-mediated regulation of transforming growth factor-β signaling in breast cancer cells. *Neoplasia* **13**, 406–418.

[197] Theodorescu D, Williams PD, Owens CR, Dziegielewski J, Moskaluk CA, Read PW, Larner JM, Story MD, Brock WA, Amundson SA, et al. (2011). Cyclophilin B expression is associated with *in vitro* radioresistance and clinical outcome after radiotherapy. *Neoplasia* **13**, 1122–1131.

[198] Tjomsland V, Spangeus A, Valila J, Sandstrom P, Borch K, Druid H, Falkmer S, Falkmer U, Messmer D, and Larsson M (2011). Interleukin 1α sustains the expression of inflammatory factors in human pancreatic cancer microenvironment by targeting cancer-associated fibroblasts. *Neoplasia* **13**, 664–675.

[199] Toyokawa G, Cho HS, Masuda K, Yamane Y, Yoshimatsu M, Hayami S, Takawa M, Iwai Y, Daigo Y, Tsuchiya E, et al. (2011). Histone lysine methyltransferase Wolf-Hirschhorn syndrome candidate 1 is involved in human carcinogenesis through regulation of the Wnt pathway. *Neoplasia* **13**, 887–898.

[200] Tseng JR, Stuart D, Aardalen K, Kaplan A, Aziz N, Hughes NP, and Gambhir SS (2011). Use of DNA microarray and small animal positron emission tomography in preclinical drug evaluation of RAF265, a novel B-Raf/VEGFR-2 inhibitor. *Neoplasia* **13**, 266–275.

[201] van der Horst G, van den Hoogen C, Buijs JT, Cheung H, Bloys H, Pelger RC, Lorenzon G, Heckmann B, Feyen J, Pujuguet P, et al. (2011). Targeting of α_v-integrins in stem/progenitor cells and supportive microenvironment impairs bone metastasis in human prostate cancer. *Neoplasia* **13**, 516–525.

[202] Vergani E, Vallacchi V, Frigerio S, Deho P, Mondellini P, Perego P, Cassinelli G, Lanzi C, Testi MA, Rivoltini L, et al. (2011). Identification of MET and SRC activation in melanoma cell lines showing primary resistance to PLX4032. *Neoplasia* **13**, 1132–1142.

[203] Volk LD, Flister MJ, Chihade D, Desai N, Trieu V, and Ran S (2011). Synergy of nab-paclitaxel and bevacizumab in eradicating large orthotopic breast tumors and preexisting metastases. *Neoplasia* **13**, 327–338.

[204] Wang H and Yan C (2011). A small-molecule p53 activator induces apoptosis through inhibiting MDMX expression in breast cancer cells. *Neoplasia* **13**, 611–619.

[205] Wang H, Zhou M, Shi B, Zhang Q, Jiang H, Sun Y, Liu J, Zhou K, Yao M, Gu J, et al. (2011). Identification of an exon 4-deletion variant of epidermal growth factor receptor with increased metastasis-promoting capacity. *Neoplasia* **13**, 461–471.

[206] Wang YW, Tu PH, Lin KT, Lin SC, Ko JY, and Jou YS (2011). Identification of oncogenic point mutations and hyperphosphorylation of anaplastic lymphoma kinase in lung cancer. *Neoplasia* **13**, 704–715.

[207] Watson GA, Naran S, Zhang X, Stang MT, Queiroz de Oliveira PE, and Hughes SJ (2011). Cytoplasmic overexpression of CD95L in esophageal adenocarcinoma cells overcomes resistance to CD95-mediated apoptosis. *Neoplasia* **13**, 198–205.

[208] Wittig-Blaich SM, Kacprzyk LA, Eismann T, Bewerunge-Hudler M, Kruse P, Winkler E, Strauss WS, Hibst R, Steiner R, Schrader M, et al. (2011). Matrix-dependent regulation of AKT in Hepsin-overexpressing PC3 prostate cancer cells. *Neoplasia* **13**, 579–589.

[209] Wu IC, Zhao Y, Zhai R, Liu G, Ter-Minassian M, Asomaning K, Su L, Liu CY, Chen F, Kulke MH, et al. (2011). Association between polymorphisms in cancer-related genes and early onset of esophageal adenocarcinoma. *Neoplasia* **13**, 386–392.

[210] Wu SY, Lan SH, Cheng DE, Chen WK, Shen CH, Lee YR, Zuchini R, and Liu HS (2011). Ras-related tumorigenesis is suppressed by BNIP3-mediated autophagy through inhibition of cell proliferation. *Neoplasia* **13**, 1171–1182.

[211] Yang F, Zhang L, Wang F, Wang Y, Huo XS, Yin YX, Wang YQ, and Sun SH (2011). Modulation of the unfolded protein response is the core of microRNA-122–involved sensitivity to chemotherapy in hepatocellular carcinoma. *Neoplasia* **13**, 590–600.

[212] Yong HY, Hwang JS, Son H, Park HI, Oh ES, Kim HH, Kim do K, Choi WS, Lee BJ, Kim HR, et al. (2011). Identification of H-Ras–specific motif for the activation of invasive signaling program in human breast epithelial cells. *Neoplasia* **13**, 98–107.

[213] Yori JL, Seachrist DD, Johnson E, Lozada KL, Abdul-Karim FW, Chodosh LA, Schiemann WP, and Keri RA (2011). Krüppel-like factor 4 inhibits tumorigenic progression and metastasis in a mouse model of breast cancer. *Neoplasia* **13**, 601–610.

[214] Zhang J, Sud S, Mizutani K, Gyetko MR, and Pienta KJ (2011). Activation of urokinase plasminogen activator and its receptor axis is essential for macrophage infiltration in a prostate cancer mouse model. *Neoplasia* **13**, 23–30.

[215] Zhang M, Herion TW, Timke C, Han N, Hauser K, Weber KJ, Peschke P, Wirkner U, Lahn M, and Huber PE (2011). Trimodal glioblastoma treatment consisting of concurrent radiotherapy, temozolomide, and the novel TGF-β receptor I kinase inhibitor LY2109761. *Neoplasia* **13**, 537–549.

[216] Adamcic U, Skowronski K, Peters C, Morrison J, and Coomber BL (2012). The effect of bevacizumab on human malignant melanoma cells with functional VEGF/VEGFR2 autocrine and intracrine signaling loops. *Neoplasia* **14**, 612–623.

[217] Ahsan A, Ramanand SG, Whitehead C, Hiniker SM, Rehemtulla A, Pratt WB, Jolly S, Gouveia C, Truong K, Van Waes C, et al. (2012). Wild-type EGFR is stabilized by direct interaction with HSP90 in cancer cells and tumors. *Neoplasia* **14**, 670–677.

[218] Al Nakouzi N, Bawa O, Le Pape A, Lerondel S, Gaudin C, Opolon P, Gonin P, Fizazi K, and Chauchereau A (2012). The IGR-CaP1 xenograft model recapitulates mixed osteolytic/blastic bone lesions observed in metastatic prostate cancer. *Neoplasia* **14**, 376–387.

[219] Al-Ansari MM, Hendrayani SF, Tulbah A, Al-Tweigeri T, Shehata AI, and Aboussekhra A (2012). $p16^{INK4A}$ represses breast stromal fibroblasts migration/invasion and their VEGF-A–dependent promotion of angiogenesis through Akt inhibition. *Neoplasia* **14**, 1269–1277.

[220] Al-Hassan NN, Behzadian A, Caldwell R, Ivanova VS, Syed V, Motamed K, and Said NA (2012). Differential roles of uPAR in peritoneal ovarian carcinomatosis. *Neoplasia* **14**, 259–270.

[221] Aschacher T, Sampl S, Kaser L, Bernhard D, Spittler A, Holzmann K, and Bergmann M (2012). The combined use of known antiviral reverse transcriptase inhibitors AZT and DDI induce anticancer effects at low concentrations. *Neoplasia* **14**, 44–53.

[222] Beghini A, Corlazzoli F, Del Giacco L, Re M, Lazzaroni F, Brioschi M, Valentini G, Ferrazzi F, Ghilardi A, Righi M, et al. (2012). Regeneration-associated WNT signaling is activated in long-term reconstituting $AC133^{bright}$ acute myeloid leukemia cells. *Neoplasia* **14**, 1236–1248.

[223] Benezra M, Hambardzumyan D, Penate-Medina O, Veach DR, Pillarsetty N, Smith-Jones P, Phillips E, Ozawa T, Zanzonico PB, Longo V, et al. (2012). Fluorine-labeled dasatinib nanoformulations as targeted molecular imaging probes in a PDGFB-driven murine glioblastoma model. *Neoplasia* **14**, 1132–1143.

[224] Blomberg Jensen M, Jorgensen A, Nielsen JE, Steinmeyer A, Leffers H, Juul A, and Rajpert-De Meyts E (2012). Vitamin D metabolism and effects on pluripotency genes and cell differentiation in testicular germ cell tumors *in vitro* and *in vivo*. *Neoplasia* **14**, 952–963.

[225] Bocci G, Fioravanti A, Orlandi P, Di Desidero T, Natale G, Fanelli G, Viacava P, Naccarato AG, Francia G, and Danesi R (2012). Metronomic ceramide analogs inhibit angiogenesis in pancreatic cancer through up-regulation of caveolin-1 and thrombospondin-1 and down-regulation of cyclin D1. *Neoplasia* **14**, 833–845.

[226] Bonezzi K, Belotti D, North BJ, Ghilardi C, Borsotti P, Resovi A, Ubezio P, Riva A, Giavazzi R, Verdin E, et al. (2012). Inhibition of SIRT2 potentiates the anti-motility activity of taxanes: implications for antineoplastic combination therapies. *Neoplasia* **14**, 846–854.

[227] Boysen G, Bausch-Fluck D, Thoma CR, Nowicka AM, Stiehl DP, Cima I, Luu VD, von Teichman A, Hermanns T, Sulser T, et al. (2012). Identification and functional characterization of pVHL-dependent cell surface proteins in renal cell carcinoma. *Neoplasia* **14**, 535–546.

[228] Briggs JW, Ren L, Nguyen R, Chakrabarti K, Cassavaugh J, Rahim S, Bulut G, Zhou M, Veenstra TD, Chen Q, et al. (2012). The ezrin metastatic phenotype is associated with the initiation of protein translation. *Neoplasia* **14**, 297–310.

[229] Buoncervello M, Borghi P, Romagnoli G, Spadaro F, Belardelli F, Toschi E, and Gabriele L (2012). Apicidin and docetaxel combination treatment drives CTCFL expression and HMGB1 release acting as potential antitumor immune response inducers in metastatic breast cancer cells. *Neoplasia* **14**, 855–867.

[230] Candolfi M, King GD, Yagiz K, Curtin JF, Mineharu Y, Muhammad AK, Foulad D, Kroeger KM, Barnett N, Josien R, et al. (2012). Plasmacytoid dendritic cells in the tumor microenvironment: immune targets for glioma therapeutics. *Neoplasia* **14**, 757–770.

[231] Cattelani S, Ferrari-Amorotti G, Galavotti S, Defferrari R, Tanno B, Cialfi S, Vergalli J, Fragliasso V, Guerzoni C, Manzotti G, et al. (2012). The p53 codon 72 Pro/Pro genotype identifies poor-prognosis neuroblastoma patients: correlation with reduced apoptosis and enhanced senescence by the p53-72P isoform. *Neoplasia* **14**, 634–643.

[232] Cekaite L, Rantala JK, Bruun J, Guriby M, Agesen TH, Danielsen SA, Lind GE, Nesbakken A, Kallioniemi O, Lothe RA, et al. (2012). MiR-9, -31, and -182 deregulation promote proliferation and tumor cell survival in colon cancer. *Neoplasia* **14**, 868–881.

[233] Cerny-Reiterer S, Ghanim V, Hoermann G, Aichberger KJ, Herrmann H, Muellauer L, Repa A, Sillaber C, Walls AF, Mayerhofer M, et al. (2012). Identification of basophils as a major source of hepatocyte growth factor in chronic myeloid leukemia: a novel mechanism of BCR-ABL1–independent disease progression. *Neoplasia* **14**, 572–584.

[234] Ceteci F, Ceteci S, Zanucco E, Thakur C, Becker M, El-Nikhely N, Fink L, Seeger W, Savai R, and Rapp UR (2012). E-cadherin controls bronchiolar progenitor cells and onset of preneoplastic lesions in mice. *Neoplasia* **14**, 1164–1177.

[235] Cho HS, Hayami S, Toyokawa G, Maejima K, Yamane Y, Suzuki T, Dohmae N, Kogure M, Kang D, Neal DE, et al. (2012). RB1 methylation by SMYD2 enhances cell cycle progression through an increase of RB1 phosphorylation. *Neoplasia* **14**, 476–486.

[236] Clark PA, Iida M, Treisman DM, Kalluri H, Ezhilan S, Zorniak M, Wheeler DL, and Kuo JS (2012). Activation of multiple ERBB family receptors mediates glioblastoma cancer stem–like cell resistance to EGFR-targeted inhibition. *Neoplasia* **14**, 420–428.

[237] Deng X, Li Q, Hoff J, Novak M, Yang H, Jin H, Erfani SF, Sharma C, Zhou P, Rabinovitz I, et al. (2012). Integrin-associated CD151 drives ErbB2-evoked mammary tumor onset and metastasis. *Neoplasia* **14**, 678–689.

[238] Derer S, Berger S, Schlaeth M, Schneider-Merck T, Klausz K, Lohse S, Overdijk MB, Dechant M, Kellner C, Nagelmeier I, et al. (2012). Oncogenic KRAS impairs EGFR antibodies' efficiency by C/EBPβ-dependent suppression of EGFR expression. *Neoplasia* **14**, 190–205.

[239] Diaz RJ, Guduk M, Romagnuolo R, Smith CA, Northcott P, Shih D, Berisha F, Flanagan A, Munoz DG, Cusimano MD, et al. (2012). High-resolution whole-genome analysis of skull base chordomas implicates FHIT loss in chordoma pathogenesis. *Neoplasia* **14**, 788–798.

[240] Domanska UM, Timmer-Bosscha H, Nagengast WB, Oude Munnink TH, Kruizinga RC, Ananias HJ, Kliphuis NM, Huls G, De Vries EG, de Jong IJ, et al. (2012). CXCR4 inhibition with AMD3100 sensitizes prostate cancer to docetaxel chemotherapy. *Neoplasia* **14**, 709–718.

[241] Fu J, Bassi DE, Zhang J, Li T, Nicolas E, and Klein-Szanto AJ (2012). Transgenic overexpression of the proprotein convertase furin enhances skin tumor growth. *Neoplasia* **14**, 271–282.

[242] Fung AS, Jonkman J, and Tannock IF (2012). Quantitative immunohistochemistry for evaluating the distribution of Ki67 and other biomarkers in tumor sections and use of the method to study repopulation in xenografts after treatment with paclitaxel. *Neoplasia* **14**, 324–334.

[243] Gadd S, Huff V, Huang CC, Ruteshouser EC, Dome JS, Grundy PE, Breslow N, Jennings L, Green DM, Beckwith JB, et al. (2012). Clinically relevant subsets identified by gene expression patterns support a revised ontogenic model of Wilms tumor: a Children's Oncology Group Study. *Neoplasia* **14**, 742–756.

[244] Gagliardi PA, di Blasio L, Orso F, Seano G, Sessa R, Taverna D, Bussolino F, and Primo L (2012). 3-Phosphoinositide–dependent kinase 1 controls breast tumor growth in a kinase-dependent but Akt-independent manner. *Neoplasia* **14**, 719–731.

[245] Galleu A, Fozza C, Simula MP, Contini S, Virdis P, Corda G, Pardini S, Cottoni F, Pruneddu S, Angeloni A, et al. (2012). $CD4^+$ and $CD8^+$ T-cell skewness in classic Kaposi sarcoma. *Neoplasia* **14**, 487–494.

[246] Gámez-Pozo A, Antón-Aparicio LM, Bayona C, Borrega P, Gallegos Sancho MI, García-Domínguez R, de Portugal T, Ramos-Vázquez M, Pérez-Carrión R, Bolós MV, et al. (2012). MicroRNA expression profiling of peripheral blood samples predicts resistance to first-line sunitinib in advanced renal cell carcinoma patients. *Neoplasia* **14**, 1144–1152.

[247] Ghazani AA, Castro CM, Gorbatov R, Lee H, and Weissleder R (2012). Sensitive and direct detection of circulating tumor cells by multimarker μ-nuclear magnetic resonance. *Neoplasia* **14**, 388–395.

[248] Glinskii OV, Sud S, Mossine VV, Mawhinney TP, Anthony DC, Glinsky GV, Pienta KJ, and Glinsky VV (2012). Inhibition of prostate cancer bone metastasis by synthetic TF antigen mimic/galectin-3 inhibitor lactulose-L-leucine. *Neoplasia* **14**, 65–73.

[249] Glogowska A, Stetefeld J, Weber E, Ghavami S, Hoang-Vu C, and Klonisch T (2012). Epidermal growth factor cytoplasmic domain affects ErbB protein degradation by the lysosomal and ubiquitin-proteasome pathway in human cancer cells. *Neoplasia* **14**, 396–409.

[250] Grund SE, Polycarpou-Schwarz M, Luo C, Eichmuller SB, and Diederichs S (2012). Rare Drosha splice variants are deficient in microRNA processing but do not affect general microRNA expression in cancer cells. *Neoplasia* **14**, 238–248.

[251] Guan B, Gao M, Wu CH, Wang TL, and Shih Ie M (2012). Functional analysis of in-frame indel ARID1A mutations reveals new regulatory mechanisms of its tumor suppressor functions. *Neoplasia* **14**, 986–993.

[252] Haeberle H, Dudley JT, Liu JT, Butte AJ, and Contag CH (2012). Identification of cell surface targets through meta-analysis of microarray data. *Neoplasia* **14**, 666–669.

[253] Hasenstein JR, Kasmerchak K, Buehler D, Hafez GR, Cleary K, Moody JS, and Kozak KR (2012). Efficacy of Tie2 receptor antagonism in angiosarcoma. *Neoplasia* **14**, 131–140.

[254] Haupenthal J, Bihrer V, Korkusuz H, Kollmar O, Schmithals C, Kriener S, Engels K, Pleli T, Benz A, Canamero M, et al. (2012). Reduced efficacy of the Plk1 inhibitor BI 2536 on the progression of hepatocellular carcinoma due to low intratumoral drug levels. *Neoplasia* **14**, 410–419.

[255] Hensley HH, Roder NA, O'Brien SW, Bickel LE, Xiao F, Litwin S, and Connolly DC (2012). Combined *in vivo* molecular and anatomic imaging for detection of ovarian carcinoma–associated protease activity and integrin expression in mice. *Neoplasia* **14**, 451–462.

[256] Hong SH, Ren L, Mendoza A, Eleswarapu A, and Khanna C (2012). Apoptosis resistance and PKC signaling: distinguishing features of high and low metastatic cells. *Neoplasia* **14**, 249–258.

[257] Hu Y, Ylivinkka I, Chen P, Li L, Hautaniemi S, Nyman TA, Keski-Oja J, and Hyytiainen M (2012). Netrin-4 promotes glioblastoma cell proliferation through integrin β4 signaling. *Neoplasia* **14**, 219–227.

[258] Jackson JB and Pallas DC (2012). Circumventing cellular control of PP2A by methylation promotes transformation in an Akt-dependent manner. *Neoplasia* **14**, 585–599.

[259] Jackson RS II, Placzek W, Fernandez A, Ziaee S, Chu CY, Wei J, Stebbins J, Kitada S, Fritz G, Reed JC, et al. (2012). Sabutoclax, a Mcl-1 antagonist, inhibits tumorigenesis in transgenic mouse and human xenograft models of prostate cancer. *Neoplasia* **14**, 656–665.

[260] Jain G, Voogdt C, Tobias A, Spindler KD, Moller P, Cronauer MV, and Marienfeld RB (2012). IκB kinases modulate the activity of the androgen receptor in prostate carcinoma cell lines. *Neoplasia* **14**, 178–189.

[261] Jaiswal AS, Armas ML, Izumi T, Strauss PR, and Narayan S (2012). Adenomatous polyposis coli interacts with flap endonuclease 1 to block its nuclear entry and function. *Neoplasia* **14**, 495–508.

[262] Jamal M, Rath BH, Tsang PS, Camphausen K, and Tofilon PJ (2012). The brain microenvironment preferentially enhances the radioresistance of $CD133^+$ glioblastoma stem-like cells. *Neoplasia* **14**, 150–158.

[263] Janakiram NB, Mohammed A, Qian L, Choi CI, Steele VE, and Rao CV (2012). Chemopreventive effects of RXR-selective rexinoid bexarotene on intestinal neoplasia of $Apc^{Min/+}$ mice. *Neoplasia* **14**, 159–168.

[264] Jarzyna PA, Deddens LH, Kann BH, Ramachandran S, Calcagno C, Chen W, Gianella A, Dijkhuizen RM, Griffioen AW, Fayad ZA, et al. (2012). Tumor angiogenesis phenotyping by nanoparticle-facilitated magnetic resonance and near-infrared fluorescence molecular imaging. *Neoplasia* **14**, 964–973.

[265] Jiang L, Greenwood TR, Artemov D, Raman V, Winnard PT Jr, Heeren RM, Bhujwalla ZM, and Glunde K (2012). Localized hypoxia results in spatially heterogeneous metabolic signatures in breast tumor models. *Neoplasia* **14**, 732–741.

[266] Judy BF, Aliperti LA, Predina JD, Levine D, Kapoor V, Thorpe PE, Albelda SM, and Singhal S (2012). Vascular endothelial-targeted therapy combined with cytotoxic chemotherapy induces inflammatory intratumoral infiltrates and inhibits tumor relapses after surgery. *Neoplasia* **14**, 352–359.

[267] Jung Y, Shiozawa Y, Wang J, McGregor N, Dai J, Park SI, Berry JE, Havens AM, Joseph J, Kim JK, et al. (2012). Prevalence of prostate cancer metastases after intravenous inoculation provides clues into the molecular basis of dormancy in the bone marrow microenvironment. *Neoplasia* **14**, 429–439.

[268] Kalyana-Sundaram S, Shankar S, Deroo S, Iyer MK, Palanisamy N, Chinnaiyan AM, and Kumar-Sinha C (2012). Gene fusions associated with recurrent amplicons represent a class of passenger aberrations in breast cancer. *Neoplasia* **14**, 702–708.

[269] Katoh H, Yamashita K, Waraya M, Margalit O, Ooki A, Tamaki H, Sakagami H, Kokubo K, Sidransky D, and Watanabe M (2012). Epigenetic silencing of HOPX promotes cancer progression in colorectal cancer. *Neoplasia* **14**, 559–571.

[270] Klingelhöfer J, Grum-Schwensen B, Beck MK, Knudsen RSP, Grigorian M, Lukanidin E, and Ambartsumian N (2012). Anti-S100A4 antibody suppresses metastasis formation by blocking stroma cell invasion. *Neoplasia* **14**, 1260–1268.

[271] Korzeniewski N, Hohenfellner M, and Duensing S (2012). CAND1 promotes PLK4-mediated centriole overduplication and is frequently disrupted in prostate cancer. *Neoplasia* **14**, 799–806.

[272] Kumbrink J and Kirsch KH (2012). Regulation of $p130^{Cas}$/BCAR1 expression in tamoxifen-sensitive and tamoxifen-resistant breast cancer cells by EGR1 and NAB2. *Neoplasia* **14**, 108–120.

[273] Kuzyk A and Mai S (2012). Selected telomere length changes and aberrant three-dimensional nuclear telomere organization during fast-onset mouse plasmacytomas. *Neoplasia* **14**, 344–351.

[274] Larbouret C, Gaborit N, Chardes T, Coelho M, Campigna E, Bascoul-Mollevi C, Mach JP, Azria D, Robert B, and Pelegrin A (2012). In pancreatic carcinoma, dual EGFR/HER2 targeting with cetuximab/trastuzumab is more effective than treatment with trastuzumab/erlotinib or lapatinib alone: implication of receptors' down-regulation and dimers' disruption. *Neoplasia* **14**, 121–130.

[275] Lecomte J, Masset A, Blacher S, Maertens L, Gothot A, Delgaudine M, Bruyere F, Carnet O, Paupert J, Illemann M, et al. (2012). Bone marrow-derived myofibroblasts are the providers of pro-invasive matrix metalloproteinase 13 in primary tumor. *Neoplasia* **14**, 943–951.

[276] Lee HJ, Yu HK, Papadopoulos JN, Kim SW, He J, Park YK, Yoon Y, Kim JS, and Kim SJ (2012). Targeted antivascular therapy with the apolipoprotein(a) kringle V, rhLK8, inhibits the growth and metastasis of human prostate cancer in an orthotopic nude mouse model. *Neoplasia* **14**, 335–343.

[277] Li J, O'Malley M, Sampath P, Kalinski P, Bartlett DL, and Thorne SH (2012). Expression of CCL19 from oncolytic vaccinia enhances immunotherapeutic potential while maintaining oncolytic activity. *Neoplasia* **14**, 1115–1121.

[278] Li Z, Owonikoko TK, Sun SY, Ramalingam SS, Doetsch PW, Xiao ZQ, Khuri FR, Curran WJ, and Deng X (2012). c-Myc suppression of DNA double-strand break repair. *Neoplasia* **14**, 1190–1202.

[279] Lin YC, Wu MH, Wei TT, Chuang SH, Chen KF, Cheng AL, and Chen CC (2012). Degradation of epidermal growth factor receptor mediates dasatinib-induced apoptosis in head and neck squamous cell carcinoma cells. *Neoplasia* **14**, 463–475.

[280] Loveless ME, Lawson D, Collins M, Nadella MV, Reimer C, Huszar D, Halliday J, Waterton JC, Gore JC, and Yankeelov TE (2012). Comparisons of the efficacy of a Jak1/2 inhibitor (AZD1480) with a VEGF signaling inhibitor (cediranib) and sham treatments in mouse tumors using DCE-MRI, DW-MRI, and histology. *Neoplasia* **14**, 54–64.

[281] Luo Y, Liu L, Rogers D, Su W, Odaka Y, Zhou H, Chen W, Shen T, Alexander JS, and Huang S (2012). Rapamycin inhibits lymphatic endothelial cell tube formation by downregulating vascular endothelial growth factor receptor 3 protein expression. *Neoplasia* **14**, 228–237.

[282] Malenda A, Skrobanska A, Issat T, Winiarska M, Bil J, Oleszczak B, Sinski M, Firczuk M, Bujnicki JM, Chlebowska J, et al. (2012). Statins impair glucose uptake in tumor cells. *Neoplasia* **14**, 311–323.

[283] Mankame TP and Lingen MW (2012). The RB tumor suppressor positively regulates transcription of the anti-angiogenic protein NOL7. *Neoplasia* **14**, 1213–1222.

[284] Maret D, Sadr MS, Sadr ES, Colman DR, Del Maestro RF, and Seidah NG (2012). Opposite roles of furin and PC5A in N-cadherin processing. *Neoplasia* **14**, 880–892.

[285] Mattei F, Schiavoni G, Sestili P, Spadaro F, Fragale A, Sistigu A, Lucarini V, Spada M, Sanchez M, Scala S, et al. (2012). IRF-8 controls melanoma progression by regulating the cross talk between cancer and immune cells within the tumor microenvironment. *Neoplasia* **14**, 1223–1235.

[286] Mohammed A, Janakiram NB, Brewer M, Duff A, Lightfoot S, Brush RS, Anderson RE, and Rao CV (2012). Endogenous n-3 polyunsaturated fatty acids delay progression of pancreatic ductal adenocarcinoma in Fat-1–p48$^{Cre/+}$–LSL-Kras$^{G12D/+}$ mice. *Neoplasia* **14**, 1249–1259.

[287] Moser C, Ruemmele P, Gehmert S, Schenk H, Kreutz MP, Mycielska ME, Hackl C, Kroemer A, Schnitzbauer AA, Stoeltzing O, et al. (2012). STAT5b as molecular target in pancreatic cancer—inhibition of tumor growth, angiogenesis, and metastases. *Neoplasia* **14**, 915–925.

[288] Mukherjee B, Tomimatsu N, Amancherla K, Camacho CV, Pichamoorthy N, and Burma S (2012). The dual PI3K/mTOR inhibitor NVP-BEZ235 is a potent inhibitor of ATM- and DNA-PKCs–mediated DNA damage responses. *Neoplasia* **14**, 34–43.

[289] Murphy AJ, de Caestecker C, Pierce J, Boyle SC, Ayers GD, Zhao Z, Libes JM, Correa H, Walter T, Huppert SS, et al. (2012). CITED1 expression in liver development and hepatoblastoma. *Neoplasia* **14**, 1153–1163.

[290] Naderi A, Meyer M, and Dowhan DH (2012). Cross-regulation between FOXA1 and ErbB2 signaling in estrogen receptor-negative breast cancer. *Neoplasia* **14**, 283–296.

[291] Ngora H, Galli UM, Miyazaki K, and Zoller M (2012). Membrane-bound and exosomal metastasis-associated C4.4A promotes migration by associating with the $\alpha_6\beta_4$ integrin and MT1-MMP. *Neoplasia* **14**, 95–107.

[292] Nord KH, Paulsson K, Veerla S, Wejde J, Brosjo O, Mandahl N, and Mertens F (2012). Retained heterodisomy is associated with high gene expression in hyperhaploid inflammatory leiomyosarcoma. *Neoplasia* **14**, 807–812.

[293] Olszewski U, Deally A, Tacke M, and Hamilton G (2012). Alterations of phosphoproteins in NCI-H526 small cell lung cancer cells involved in cytotoxicity of cisplatin and titanocene Y. *Neoplasia* **14**, 813–822.

[294] Paulo P, Ribeiro FR, Santos J, Mesquita D, Almeida M, Barros-Silva JD, Itkonen H, Henrique R, Jeronimo C, Sveen A, et al. (2012). Molecular subtyping of primary prostate cancer reveals specific and shared target genes of different ETS rearrangements. *Neoplasia* **14**, 600–611.

[295] Qiu X, Guo G, Chen K, Kashiwada M, Druker BJ, Rothman PB, and Chen JL (2012). A requirement for SOCS-1 and SOCS-3 phosphorylation in Bcr-Abl–induced tumorigenesis. *Neoplasia* **14**, 547–558.

[296] Rahman M, Selvarajan K, Hasan MR, Chan AP, Jin C, Kim J, Chan SK, Le ND, Kim YB, and Tai IT (2012). Inhibition of COX-2 in colon cancer modulates tumor growth and MDR-1 expression to enhance tumor regression in therapy-refractory cancers *in vivo*. *Neoplasia* **14**, 624–633.

[297] Rao CV, Mohammed A, Janakiram NB, Li Q, Ritchie RL, Lightfoot S, Vibhudutta A, and Steele VE (2012). Inhibition of pancreatic intraepithelial neoplasia progression to carcinoma by nitric oxide-releasing aspirin in p48$^{Cre/+}$–LSL-Kras$^{G12D/+}$ mice. *Neoplasia* **14**, 778–787.

[298] Reiner T, Lacy J, Keliher EJ, Yang KS, Ullal A, Kohler RH, Vinegoni C, and Weissleder R (2012). Imaging therapeutic PARP inhibition *in vivo* through bioorthogonally developed companion imaging agents. *Neoplasia* **14**, 169–177.

[299] Ronchi CL, Leich E, Sbiera S, Weismann D, Rosenwald A, Allolio B, and Fassnacht M (2012). Single nucleotide polymorphism microarray analysis in cortisol-secreting adrenocortical adenomas identifies new candidate genes and pathways. *Neoplasia* **14**, 206–218.

[300] Samanta D, Kaufman J, Carbone DP, and Datta PK (2012). Long-term smoking mediated down-regulation of Smad3 induces resistance to carboplatin in non–small cell lung cancer. *Neoplasia* **14**, 644–655.

[301] Shang X, Lin X, Alvarez E, Manorek G, and Howell SB (2012). Tight junction proteins claudin-3 and claudin-4 control tumor growth and metastases. *Neoplasia* **14**, 974–985.

[302] Soria G, Lebel-Haziv Y, Ehrlich M, Meshel T, Suez A, Avezov E, Rozenberg P, and Ben-Baruch A (2012). Mechanisms regulating the secretion of the pro-malignancy chemokine CCL5 by breast tumor cells: CCL5's 40s loop and intracellular glycosaminoglycans. *Neoplasia* **14**, 1–19.

[303] Stigliani S, Coco S, Moretti S, Oberthuer A, Fischer M, Theissen J, Gallo F, Garavent A, Berthold F, Bonassi S, et al. (2012). High genomic instability predicts survival in metastatic high-risk neuroblastoma. *Neoplasia* **14**, 823–832.

[304] Sun X, Essalmani R, Day R, Khatib AM, Seidah NG, and Prat AP (2012). Proprotein convertase subtilisin/kexin type 9 deficiency reduces melanoma metastasis in liver. *Neoplasia* **14**, 1122–1131.

[305] Tanikawa C, Nakagawa H, Furukawa Y, Nakamura Y, and Matsuda K (2012). CLCA2 as a p53-inducible senescence mediator. *Neoplasia* **14**, 141–149.

[306] Taniuchi K, Yokotani K, and Saibara T (2012). BART inhibits pancreatic cancer cell invasion by Rac1 inactivation through direct binding to active Rac1. *Neoplasia* **14**, 440–450.

[307] Thomasova D, Mulay SR, Bruns H, and Anders HJ (2012). p53-Independent roles of MDM2 in NF-κB signaling: Implications for cancer therapy, wound healing, and autoimmune diseases. *Neoplasia* **14**, 1097–1101.

[308] Treviño JG, Pillai S, Kunigal S, Singh S, Fulp WJ, Centeno BA, and Chellappan SP (2012). Nicotine induces inhibitor of differentiation-1 in a Src-dependent pathway promoting metastasis and chemoresistance in pancreatic adenocarcinoma. *Neoplasia* **14**, 1102–1114.

[309] Trivigno D, Essmann F, Huber SM, and Rudner J (2012). Deubiquitinase USP9x confers radioresistance through stabilization of Mcl-1. *Neoplasia* **14**, 893–904.

[310] Venkatesha VA, Parsels LA, Parsels JD, Zhao L, Zabludoff SD, Simeone DM, Maybaum J, Lawrence TS, and Morgan MA (2012). Sensitization of pancreatic cancer stem cells to gemcitabine by Chk1 inhibition. *Neoplasia* **14**, 519–525.

[311] Volk-Draper LD, Rajput S, Hall KL, Wilber A, and Ran S (2012). Novel model for basaloid triple-negative breast cancer: behavior *in vivo* and response to therapy. *Neoplasia* **14**, 926–942.

[312] von dem Knesebeck A, Felsberg J, Waha A, Hartmann W, Scheffler B, Glas M, Hammes J, Mikeska T, Yan PS, Endl E, et al. (2012). RANK (TNFRSF11A) is epigenetically inactivated and induces apoptosis in gliomas. *Neoplasia* **14**, 526–534.

[313] Wang R, Asangani IA, Chakravarthi BV, Ateeq B, Lonigro RJ, Cao Q, Mani RS, Camacho DF, McGregor N, Schumann TE, et al. (2012). Role of transcriptional corepressor CtBP1 in prostate cancer progression. *Neoplasia* **14**, 905–914.

[314] Wang Z, Zhong J, Inuzuka H, Gao D, Shaik S, Sarkar FH, and Wei W (2012). An evolving role for DEPTOR in tumor development and progression. *Neoplasia* **14**, 368–375.

[315] Weekes CD, Song D, Arcaroli J, Wilson LA, Rubio-Viqueira B, Cusatis G, Garrett-Mayer E, Messersmith WA, Winn RA, and Hidalgo M (2012). Stromal cell–derived factor 1α mediates resistance to mTOR-directed therapy in pancreatic cancer. *Neoplasia* **14**, 690–701.

[316] Woldemichael GM, Turbyville TJ, Vasselli JR, Linehan WM, and McMahon JB (2012). Lack of a functional VHL gene product sensitizes renal cell carcinoma

cells to the apoptotic effects of the protein synthesis inhibitor verrucarin A. *Neoplasia* **14**, 771–777.

[317] Wong HK, Shimizu A, Kirkpatrick ND, Garkavtsev I, Chan AW, di Tomaso E, Klagsbrun M, and Jain RK (2012). Merlin/NF2 regulates angiogenesis in schwannomas through a Rac1/semaphorin 3F–dependent mechanism. *Neoplasia* **14**, 84–94.

[318] Wu X, Tao Y, Hou J, Meng X, and Shi J (2012). Valproic acid upregulates NKG2D ligand expression through an ERK-dependent mechanism and potentially enhances NK cell–mediated lysis of myeloma. *Neoplasia* **14**, 1178–1189.

[319] Yamashita S, Lai KP, Chuang KL, Xu D, Miyamoto H, Tochigi T, Pang ST, Li L, Arai Y, Kung HJ, et al. (2012). ASC-J9 suppresses castration-resistant prostate cancer growth through degradation of full-length and splice variant androgen receptors. *Neoplasia* **14**, 74–83.

[320] Yang HW, Kim TM, Song SS, Shrinath N, Park R, Kalamarides M, Park PJ, Black PM, Carroll RS, and Johnson MD (2012). Alternative splicing of CHEK2 and codeletion with NF2 promote chromosomal instability in meningioma. *Neoplasia* **14**, 20–28.

[321] Yang Y, Jiang H, Gao H, Kong J, Zhang P, Hu S, Shi B, Yao M, and Li Z (2012). The monoclonal antibody CH12 enhances the sorafenib-mediated growth inhibition of hepatocellular carcinoma xenografts expressing epidermal growth factor receptor variant III. *Neoplasia* **14**, 509–518.

[322] Yu L, Tumati V, Tseng SF, Hsu FM, Kim DN, Hong D, Hsieh JT, Jacobs C, Kapur P, and Saha D (2012). DAB2IP regulates autophagy in prostate cancer in response to combined treatment of radiation and a DNA-PKcs inhibitor. *Neoplasia* **14**, 1203–1212.

[323] Zhai R, Zhao Y, Su L, Cassidy L, Liu G, and Christiani DC (2012). Genome-wide DNA methylation profiling of cell-free serum DNA in esophageal adenocarcinoma and Barrett esophagus. *Neoplasia* **14**, 29–33.

[324] Zhao Y and Sun Y (2012). Targeting the mTOR-DEPTOR pathway by CRL E3 ubiquitin ligases: therapeutic application. *Neoplasia* **14**, 360–367.