## Errata

Volume 32, No. 12, page 5281, right column: Line 16 should read:

control group (Figure 4A). After treatment of 100 nM DTX-ĸ

Volume 32, No. 12, page 5283-4: The section of References should read:

## References

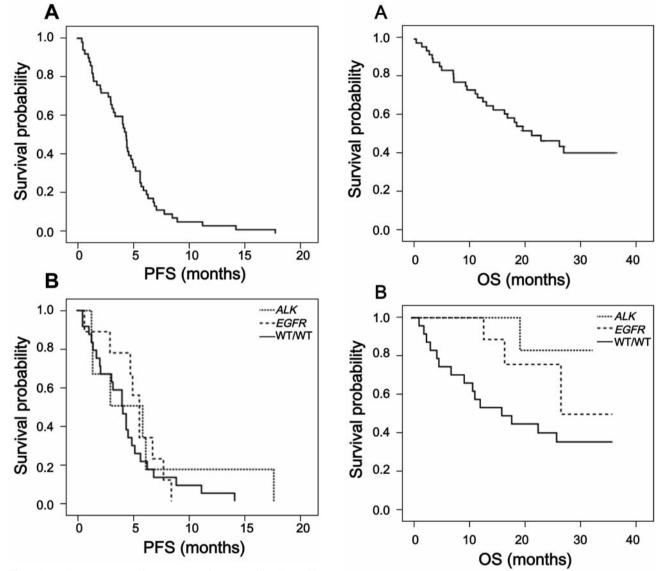
- 1 Miller C: An overview of the potassium channel family. Genome Biol 1: REVIEWS0004.1-0004.5, 2000.
- 2 Shieh CC, Coghlan M, Sullivan JP and Gopalakrishnan M: Potassium channels: molecular defects, diseases, and therapeutic opportunities. Pharmacol Rev 52: 557-594, 2000.
- 3 Edwards G and Weston AH: The role of potassium channels in excitable cells. Diabetes Res Clin Pract 28 Suppl: S57-66, 1995.
- 4 O'Grady SM and Lee SY: Molecular diversity and function of voltage-gated (Kv) potassium channels in epithelial cells. Int J Biochem Cell Biol 37: 1578-1594, 2005.
- 5 Pongs O: Voltage-gated potassium channels: from hyperexcitability to excitement. FEBS Lett 452: 31-35, 1999.
- 6 Boland LM and Jackson KA: Protein kinase C inhibits Kv1.1 potassium channel function. Am J Physiol 277: C100-110, 1999.
- 7 Dubois JM and Rouzaire-Dubois B: Role of potassium channels in mitogenesis. Prog Biophys Mol Biol 59: 1-21, 1993.
- 8 Kunzelmann K: Ion channels and cancer. J Membr Biol 205: 159-173, 2005.
- 9 Pardo LA: Voltage-gated potassium channels in cell proliferation. Physiology (Bethesda) 19: 285-292, 2004.
- 10 Pardo LA, Contreras-Jurado C, Zientkowska M, Alves F and Stuhmer W: Role of voltage-gated potassium channels in cancer. J Membr Biol 205: 115-124, 2005.
- 11 Camacho J: Ether a go-go potassium channels and cancer. Cancer Lett 233: 1-9, 2006.
- 12 Wonderlin WF and Strobl JS: Potassium channels, proliferation and G1 progression. J Membr Biol 154: 91-107, 1996.
- 13 Wang Z: Roles of K<sup>+</sup> channels in regulating tumour cell proliferation and apoptosis. Pflugers Arch *448*: 274-286, 2004.
- 14 Jang SH, Choi SY, Ryu PD and Lee SY: Anti-proliferative effect of Kv1.3 blockers in A549 human lung adenocarcinoma *in vitro* and *in vivo*. Eur J Pharmacol 651: 26-32, 2011.
- 15 Jang SH, Ryu PD and Lee SY: Dendrotoxin-kappa suppresses tumor growth induced by human lung adenocarcinoma A549 cells in nude mice. J Vet Sci 12: 35-40, 2011.
- 16 Zhou Q, Kwan HY, Chan HC, Jiang JL, Tam SC and Yao X: Blockage of voltage-gated K<sup>+</sup> channels inhibits adhesion and proliferation of hepatocarcinoma cells. Int J Mol Med 11: 261-266, 2003.
- 17 Rouzaire-Dubois B and Dubois JM: K<sup>+</sup> channel block-induced mammalian neuroblastoma cell swelling: A possible mechanism to influence proliferation. J Physiol 510(Pt 1): 93-102, 1998.
- 18 Jang SH, Kang KS, Ryu PD and Lee SY: Kv1.3 voltage-gated K(+) channel subunit as a potential diagnostic marker and therapeutic target for breast cancer. BMB Rep 42: 535-539, 2009.

- 19 Jang SH, Choi C, Hong SG, Yarishkin OV, Bae YM, Kim JG, O'Grady SM, Yoon KA, Kang KS, Ryu PD and Lee SY: Silencing of Kv4.1 potassium channels inhibits cell proliferation of tumorigenic human mammary epithelial cells. Biochem Biophys Res Commun 384: 180-186, 2009.
- 20 Kim HJ, Jang SH, Jeong YA, Ryu PD, Kim DY and Lee SY: Involvement of Kv4.1 K(+) channels in gastric cancer cell proliferation. Biol Pharm Bull *33*: 1754-1757, 2010.
- 21 Harvey AL: Recent studies on dendrotoxins and potassium ion channels. Gen Pharmacol 28: 7-12, 1997.
- 22 Smith LA, Reid PF, Wang FC, Parcej DN, Schmidt JJ, Olson MA and Dolly JO: Site-directed mutagenesis of dendrotoxin K reveals amino acids critical for its interaction with neuronal K<sup>+</sup> channels. Biochemistry 36: 7690-7696, 1997.
- 23 Wang FC, Bell N, Reid P, Smith LA, McIntosh P, Robertson B and Dolly JO: Identification of residues in dendrotoxin K responsible for its discrimination between neuronal K<sup>+</sup> channels containing Kv1.1 and 1.2 alpha subunits. Eur J Biochem 263: 222-229, 1999.
- 24 Armour AA and Watkins CL: The challenge of targeting EGFR: experience with gefitinib in nonsmall cell lung cancer. Eur Respir Rev 19: 186-196, 2010.
- 25 Sahu RP, Batra S, Kandala PK, Brown TL and Srivastava SK: The role of *K-ras* gene mutation in TRAIL-induced apoptosis in pancreatic and lung cancer cell lines. Cancer Chemother Pharmacol 67: 481-487, 2011.
- 26 Furugaki K, Iwai T, Shirane M, Kondoh K, Moriya Y and Mori K: Schedule-dependent antitumor activity of the combination with erlotinib and docetaxel in human non-small cell lung cancer cells with *EGFR* mutation, *KRAS* mutation or both wild-type *EGFR* and *KRAS*. Oncol Rep 24: 1141-1146, 2010.
- 27 Rodenhuis S, Slebos RJ, Boot AJ, Evers SG, Mooi WJ, Wagenaar SS, van Bodegom PC and Bos JL: Incidence and possible clinical significance of *K-ras* oncogene activation in adenocarcinoma of the human lung. Cancer Res 48: 5738-5741, 1988.
- 28 Suzuki Y, Orita M, Shiraishi M, Hayashi K and Sekiya T: Detection of *RAS* gene mutations in human lung cancers by single-strand conformation polymorphism analysis of polymerase chain reaction products. Oncogene 5: 1037-1043, 1990.
- 29 Rodenhuis S and Slebos RJ: The RAS oncogenes in human lung cancer. Am Rev Respir Dis 142: S27-30, 1990.
- 30 Janmaat ML, Rodriguez JA, Gallegos-Ruiz M, Kruyt FA and Giaccone G: Enhanced cytotoxicity induced by gefitinib and specific inhibitors of the Ras or phosphatidyl inositol-3 kinase pathways in non-small cell lung cancer cells. Int J Cancer 118: 209-214, 2006.
- 31 Seshacharyulu P, Ponnusamy MP, Haridas D, Jain M, Ganti AK and Batra SK: Targeting the EGFR signaling pathway in cancer therapy. Expert Opin Ther Targets 16: 15-31, 2012.

- 32 Goldie JH and Coldman AJ: The genetic origin of drug resistance in neoplasms: implications for systemic therapy. Cancer Res 44: 3643-3653, 1984.
- 33 Park IH, Kim JY, Jung JI and Han JY: Lovastatin overcomes gefitinib resistance in human non-small cell lung cancer cells with *K-Ras* mutations. Invest New Drugs 28: 791-799, 2010.
- 34 Bill A, Schmitz A, Konig K, Heukamp LC, Hannam JS and Famulok M: Anti-proliferative effect of cytohesin inhibition in gefitinib-resistant lung cancer cells. PLoS One 7: e41179, 2012.
- 35 Ouadid-Ahidouch H, Chaussade F, Roudbaraki M, Slomianny C, Dewailly E, Delcourt P and Prevarskaya N: KV1.1 K(+) channels identification in human breast carcinoma cells: involvement in cell proliferation. Biochem Biophys Res Commun 278: 272-277, 2000.
- 36 Pao W, Miller V, Zakowski M, Doherty J, Politi K, Sarkaria I, Singh B, Heelan R, Rusch V, Fulton L, Mardis E, Kupfer D, Wilson R, Kris M and Varmus H: EGF receptor gene mutations are common in lung cancers from "never smokers" and are associated with sensitivity of tumors to gefitinib and erlotinib. Proc Natl Acad Sci USA 101: 13306-13311, 2004.
- 37 Sordella R, Bell DW, Haber DA and Settleman J: Gefitinibsensitizing EGFR mutations in lung cancer activate antiapoptotic pathways. Science 305: 1163-1167, 2004.
- 38 Eberhard DA, Johnson BE, Amler LC, Goddard AD, Heldens SL, Herbst RS, Ince WL, Janne PA, Januario T, Johnson DH, Klein P, Miller VA, Ostland MA, Ramies DA, Sebisanovic D, Stinson JA, Zhang YR, Seshagiri S and Hillan KJ: Mutations in the epidermal growth factor receptor and in *KRAS* are predictive and prognostic indicators in patients with non-small cell lung cancer treated with chemotherapy alone and in combination with erlotinib. J Clin Oncol 23: 5900-5909, 2005.

- 39 Massarelli E, Varella-Garcia M, Tang X, Xavier AC, Ozburn NC, Liu DD, Bekele BN, Herbst RS and Wistuba II: *KRAS* mutation is an important predictor of resistance to therapy with epidermal growth factor receptor tyrosine kinase inhibitors in non-smallcell lung cancer. Clin Cancer Res 13: 2890-2896, 2007.
- 40 Giaccone G and Wang Y: Strategies for overcoming resistance to EGFR family tyrosine kinase inhibitors. Cancer Treat Rev *37*: 456-464, 2011.
- 41 Petrelli A and Giordano S: From single- to multi-target drugs in cancer therapy: when aspecificity becomes an advantage. Curr Med Chem *15*: 422-432, 2008.
- 42 Faivre S, Delbaldo C, Vera K, Robert C, Lozahic S, Lassau N, Bello C, Deprimo S, Brega N, Massimini G, Armand JP, Scigalla P and Raymond E: Safety, pharmacokinetic, and antitumor activity of SU11248, a novel oral multitarget tyrosine kinase inhibitor, in patients with cancer. J Clin Oncol 24: 25-35, 2006.
- 43 Zimmermann GR, Lehar J and Keith CT: Multi-target therapeutics: when the whole is greater than the sum of the parts. Drug Discov Today *12*: 34-42, 2007.
- 44 Zhang DY, Zhang YH, Sun HY, Lau CP and Li GR: Epidermal growth factor receptor tyrosine kinase regulates the human inward rectifier potassium K(IR)2.3 channel, stably expressed in HEK 293 cells. Br J Pharmacol *164*: 1469-1478, 2011.
- 45 Bowlby MR, Fadool DA, Holmes TC and Levitan IB: Modulation of the Kv1.3 potassium channel by receptor tyrosine kinases. J Gen Physiol *110*: 601-610, 1997.

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Volume 33, No. 8, page 3330: Figures 1 and 2 should read:

Figure 1. Kaplan-Meier curves for progression-free survival (PFS) (N=50). The median PFS (N=50) was 4.3 months (A). The median PFS in subgroups of with patients with anaplastic lymphoma kinase (ALK) translocation, epidermal growth factor receptor (EGFR) mutation, wild-type for both ALK and EGFR (WT/WT) were 3.0, 5.5 and 4.0 months, respectively (B).

Figure 2. Kaplan–Meier curves for overall survival (OS) (N=50). The overall median OS (N=50) was 22.2 months (A). The median OS had not yet been reached in the patients with epidermal growth factor receptor (EGFR) mutations or anaplastic lymphoma kinase (ALK) translocation, but was 15.8 months in wild-type for both ALK and EGFR (WT/WT) patients (B).

Volume 33, No. 8, page 3462: The last two lines of Table I should read:

3-Year	44.3
5-Year	34.3

## Volume 33, No. 8, page 3463: Table II should be replaced with:

Variable	Univariate <i>p</i> -value <sup>1</sup>		Multivariate <sup>2</sup>		
		HR	95% CI	<i>p</i> -value <sup>3</sup>	
Gender					
Male vs. female	0.0561	-	-	-	
Age					
<67 <i>vs.</i> >68	0.3494	-	-	-	
Approach					
Abdominal vs. thoracotomy	0.1276	-	-	-	
Histological type					
Undifferentiated and others vs. differentiated	0.3904	-	-	-	
Macroscopic type					
Type 3-5 <i>vs</i> . type 0-2	0.0018	-	-	-	
Tumor depth					
pT3-4 vs. pT1-2	0.0513	-	-	-	
pT4 vs. pT1-3	0.0049	-	-	-	
Lymph node involvement					
Positive vs. negative	0.0001	12.9270	1.512-110.533	0.0194	
Lymphatic invasion					
Positive vs. negative	0.0077	-	-	-	
Histological venous invasion					
Positive vs. negative	0.7160	-	-	-	

Table II. Cox proportional hazard regression analysis for overall survival in 47 consecutive patients with Siewert type II adenocarcinoma.

<sup>1</sup>Univariate analysis using the log-rank test. <sup>2</sup>Cox proportional hazard regression analysis for overall survival. Considered significant at 0.05. HR, Hazard ratio; CI, 95% confidence interval.

Volume 33, No. 8, page 3463: Left column, lines 4 and 5 should read:

group of patients with tumor depth pT1-3 (n=24, 5-year overall survival rate; 51%) than in the group of patients with tumor depth pT4 (n=23, 5-year overall survival rate; 49%)

Volume 33, No. 8, page 3464: The legend of Figure 1 should read:

Figure 1. Overall survival rates after surgery of Siewert type II adenocarcinoma (n=47). The 5-year overall survival rate is significantly better in the group of patients with tumor depth pT1-3 (n=24, 5-year overall survival rate; 51%) than in the group of patients with tumor depth pT4 (n=23, 5-year overall survival rate; 49%) (p=0.0049).

Volume 33, No. 8, page 3481: The name of the 4th author should read:

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