Correction

In the publication of this article [1], there was an error in the Methods, Cell cultures and reagents section: The Methods, Cell cultures and reagents section: The following primary antibodies were used: P62 (#8025), phospho-H2AX(γ-H2AX; #9718), caspase-8 (#9746), RIP1 (#3493s), Beclin1 (#3738s), ATG7 (#2631S), PARP (#9546S), caspase-3 (#9665s), cIAP1 (#3493s), cIAP2 (#3130s), XIAP (#14334), FADD (#2782S), phospho-NF-kBp105/p50 (#4806S), NF-κBp100/p52 (#3845S), TNF-α ( #6945s), TNF-α neutralizing antibody ( #7321s), and TNFR1 (#3736S) were purchased from Cell Signaling Technology Inc.; GAPDH (#sc-47724) was from Santa Cruz Biotechnology (SC); LC3 (#NB100–2220) was from Novus Biologicals. Z-VAD-FMK (#V116) and Necrostatin-1 (#N9037) were from Sigma. IKK-16 (#S2882) from Selleck.

In the Results, APG-1387 is RIP1-dependent in ovarian cancer induced apoptosis section there was an error: ‘We examined the protein levels of caspase-8/RIP1 by western blot, as shown in Fig. 4a’.

Should instead read: ‘We examined the protein levels of caspase-8/RIP1 by western blot. APG-1387 triggered the activation of caspase-8 and downregulated the protein level of RIP1, as shown in Fig. 4a’.

In the Results, APG-1387 induces apoptotic cell death through engagement of TNFR1 by TNF-alpha signaling pathway section there was an error: ‘We have investigated the expression of NF-κB1/p50 and NF-κB2/p52 by

© The Author(s). 2018 Open Access This article is distributed under the terms of the Creative Commons Attribution 4.0 International License (http://creativecommons.org/licenses/by/4.0/), which permits unrestricted use, distribution, and reproduction in any medium, provided you give appropriate credit to the original author(s) and the source, provide a link to the Creative Commons license, and indicate if changes were made. The Creative Commons Public Domain Dedication waiver (http://creativecommons.org/publicdomain/zero/1.0/) applies to the data made available in this article, unless otherwise stated.
western blot after cells were incubated with various concentrations of APG-1387'.

Should instead read: ‘These results demonstrate that TNFα signaling is required for APG-1387-induced apoptotic cell death. Next, we have investigated the expression of NF-κB1/p50 and NF-κB2/p52 by western blot after cells were incubated with various concentrations of APG-1387’.

A section header contained an error: ‘APG-1387-induced autophagy’

Should instead read: ‘APG-1387 induces autophagy in ovarian cancer’

A section header contained an error: ‘APG-1387-induced apoptosis by modulating autophagy’

Should instead read: ‘Inhibition of autophagy sensitizes ovarian cancer cells to APG-1387-induced apoptosis’.

In the Discussion section there was an error: ‘Our results suggest that APG-1387 induces autophagy during apoptosis. Autophagy plays a role in protecting cell survival. We have also found that it was effective as a single agent in vivo models. Treatment with APG-1387 induced potent cytotoxic and anti-proliferative activity against established and human ovarian cancer cells’.

Should instead read: ‘Our results suggest that APG-1387 induces autophagy while triggering apoptosis. APG-1387-induced autophagy plays a role in protecting cell survival and inhibition of autophagy potentiates cytotoxicity of APG-1387 in ovarian cancer cells’.

Figure 7 caption had an error: ‘APG-1387-induced autophagy promotes cell death in SKOV3 cells’.

Should instead read: ‘Fig. 7 APG-1387-induced autophagy promotes cell survival in SKOV3 cells’.

In the Funding section there was an error due to the fact some additional funds were missing.

Should instead additionally read: ‘the Project of International Science and Technology Cooperation of Fujian Province (Grant number. 2015I0002); and the Joint Funds for the innovation of science and Technology, Fujian province (Grant number. 2016Y91030065). The correct Figs. 2, 3 and 6 are presented here:

This has now been updated in the original article [1].
Fig. 2 Effects of APG-1387 on apoptosis in ovarian cancer. a APG-1387 inhibited the proliferation of SKOV3 cell line. They were treated with the indicated concentrations of APG-1387 for 24, 48, 72 h. Cell viability was determined by the CCK-8 assay. b Morphology of SKOV3 cells exposed to APG-1387 (0, 10 nM) photographed under a fluorescence microscope (original magnification × 10). c APG-1387-induced apoptosis in SKOV3 cells was assessed by Hoechst33258 staining. Morphology of SKOV3 cells exposed to APG-1387 at different concentrations photographed under a fluorescence microscope (original magnification × 10). Condensed and fragmented nuclei were the mean ± SEM of 5 randomized areas. P < 0.01. d SKOV3 cells were treated with 10 nM APG-1387 for the indicated times. The cells were stained for phosphorylated H2AX and DAPI, then were analyzed by fluorescence microscopy (original magnification × 200). γ-H2AX positive spots were the mean ± SEM of 5 randomized areas. P < 0.01. e, f SKOV3 and OVCAR3 cells were exposed to various concentrations of APG-1387 (0, 10, 30 nM) for 24 h followed cell apoptosis analysis by flow cytometry. g Western blot analysis of caspase-3/PARP SKOV3 cells were treated with APG-1387 (0, 3, 10, 30, 100, 300 nM) for 24 h. The data shown are representative of three different experiments. h SKOV3 cells were stimulated with APG-1387 for indicated periods of concentrations, caspase activation were tested by caspase activity assay.
**Fig. 3** APG-1387-induced apoptosis in caspase dependent manner. 

- **a** Cells with or without addition of Z-VAD-FMK. Morphology of cells exposed to different treatment groups photographed under a fluorescence microscope (original magnification × 10).

- **b** APG-1387 was coadministered with or without addition of caspase inhibitor (Z-VAD-FMK). Cell viability was determined by the CCK-8 assay.

- **c** Western blot analysis of the effect of APG-1387 with or without addition of Z-VAD-FMK on caspase-3/PARP expression level in SKOV3 cells.

- **d** Western blot analysis of the expression levels of IAPs at different concentrations of APG-1387 in SKOV3 cells. Cells were treated with different time points, and the effect of APG-1387 on IAP family members expression level was determined by western blot. Data represent one of three experiments yielding similar results.
Fig. 6 APG-1387 induces autophagy in ovarian cancer cells. a The expression of LC3, Beclin1 and P62 was measured by western blot. Cells were treated with APG-1387(0, 3, 10, 30, 100, 300 nM) for 24 h. b Cells were transfected with GFP-LC3 plasmids, and then maintained in media with or without 3 nM APG-1387 for 24 h. The cells were then stained with DAPI and analyzed by fluorescence microscopy. c Statistical analysis of the percentage of LC3 puncta per cell. Columns, mean (n = 3); bars, SD. *P < 0.01 vs. untreated group. LC3 puncta per cell were quantified. d Cells were transfected with Beclin1 siRNAs. Western blot was used to detect the expression of Beclin1. e Cells were transfected with ATG7 siRNAs. Western blot was used to detect the expression of ATG7. f Cells were transfected with Beclin1 siRNAs. After 24 h treatment with or without 3 nM APG-1387, western blot analysis was performed for indicated proteins. g Cells were transfected with ATG7 siRNAs. After 24 h treatment with or without 3 nM APG-1387, western blot analysis was performed for indicated proteins. h Western blot analysis was performed for indicated proteins in cells transfected with siBeclin-1 and treated with 10 nM APG-1387. i Western blot analysis was performed for indicated proteins in cells transfected with siATG7-1 and treated with 10 nM APG-1387.
Author details
1State Key Laboratory of Oncology in South China, Collaborative Innovation Center for Cancer Medicine, Sun Yat-sen University Cancer Center, Guangzhou 510060, China. 2Department of Pharmacology, Fujian Provincial Key Laboratory of Natural Medicine Pharmacology, School of Pharmacy, Fujian Medical University, Fuzhou 350108, China. 3Ascentage Pharma Group Corp., Ltd, Taizhou 225309, China. 4Department of Medical Oncology, State Key Laboratory of Oncology in South China, Collaborative Innovation Center for Cancer Medicine, Sun Yat-Sen University Cancer Center, Guangzhou 510060, China. 5Departments of Clinical Laboratory, State Key Laboratory of Oncology in South China, Collaborative Innovation Center for Cancer Medicine, Sun Yat-Sen University Cancer Center, 651 Dongfeng Road East, Guangzhou 510060, China.

Received: 14 March 2018 Accepted: 14 March 2018
Published online: 24 May 2018

Reference
1. Li BX, Wang HB, Qiu MZ, et al. Novel smac mimetic APG-1387 elicits ovarian cancer cell killing through TNF-alpha, Ripoptosome and autophagy mediated cell death pathway. J Exp Clin Cancer Res. 2018;37:53. https://doi.org/10.1186/s13046-018-0703-9