

Correction

Correction: Wogonoside prevents colitis-associated colorectal carcinogenesis and colon cancer progression in inflammation-related microenvironment via inhibiting NF- κ B activation through PI3K/Akt pathway

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Published:

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This article has been corrected: During the preparation of Figure 6, the incorrect Ki67 assay results were accidentally used. The correct Ki67 assay images in Figure 6 are shown below. The authors declare that these corrections do not change the results or conclusions of this paper.

Original article: Oncotarget. 2016; 7:34300–34315. <https://doi.org/10.18632/oncotarget.8815>

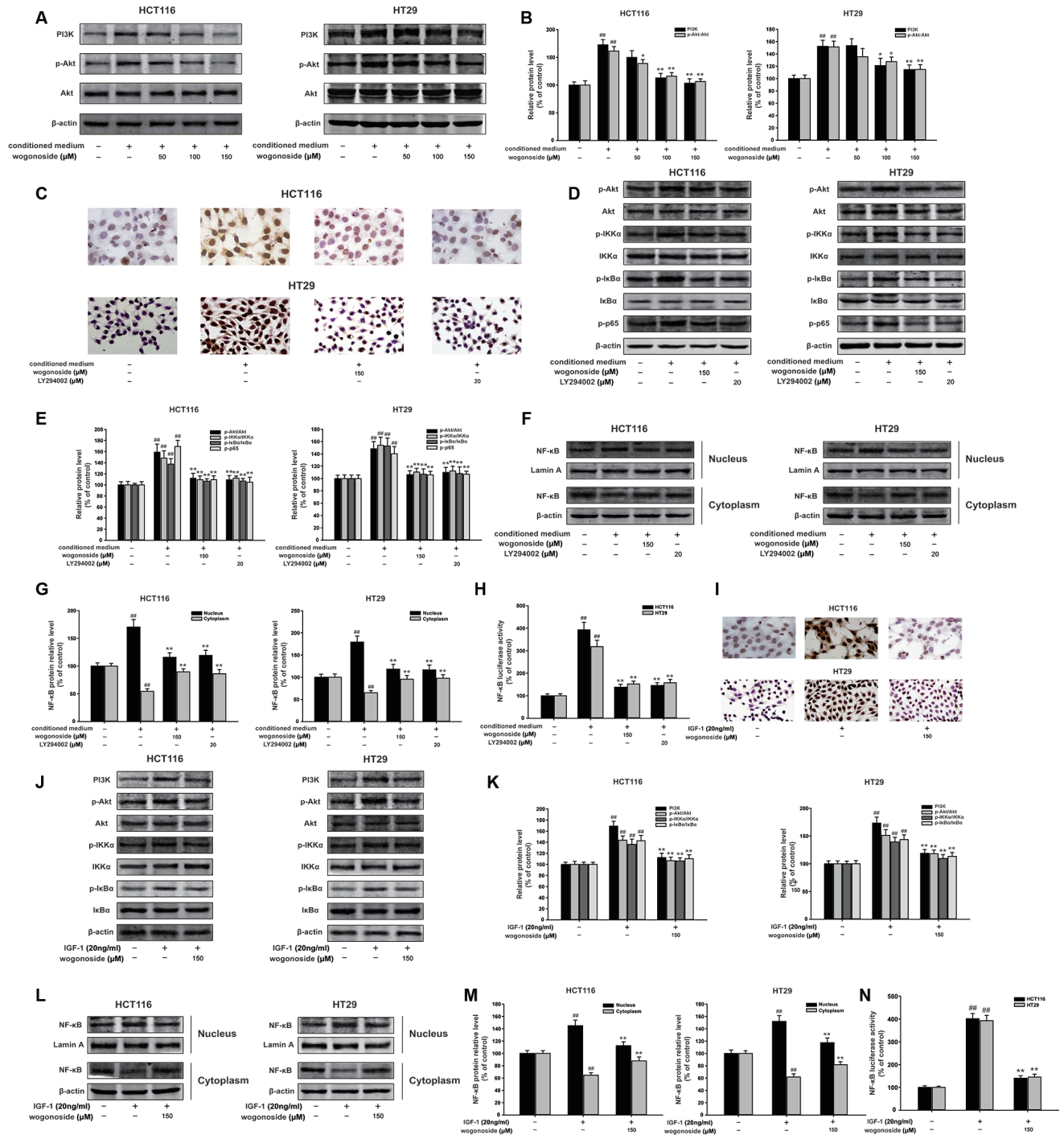


Figure 6: Wogonoside inhibited the proliferation of human colon cancer cells exposed to the conditioned media from LPS-activated THP-1 cells via inhibition of NF-κB activation through PI3K/Akt pathway. (A) The protein expression of PI3K, p-Akt, Akt in HCT116 and HT29 cells in all groups was analyzed by Western Blot. (B) Densitometric analysis to determine the relative ratio normalized to β-actin. The results are representative of three independent experiments and expressed as mean ± SD. ##P < 0.01 compared with control group; *P < 0.05, **P < 0.01 compared with conditioned media group. (C–H) HCT116 and HT29 cells were cultured in the conditional culture system with wogonoside (150 μM) or LY294002 (20 μM) for 24 h. (C) Ki67 cell proliferation detection of HCT-116 and HT29 cells in the conditional culture system treated with wogonoside or LY294002. (D) The protein expression of p-Akt,

Akt, p-IKK α , IKK α , p-I κ B α , I κ B α and p-p65 was analyzed by Western Blot. **(E)** Densitometric analysis to determine the relative ratio normalized to β -actin. The results are representative of three independent experiments and expressed as mean \pm SD, ## P<0.01 compared with control group; **P<0.01 compared with conditioned media group. **(F)** NF- κ B p65 nuclear translocation in HCT116 and HT29 cells in all group were determined by Western Blot. Wogonoside inhibited the proliferation of human colon cancer cells exposed to the conditioned media from LPS-activated THP-1 cells via inhibition of NF- κ B activation through PI3K/Akt pathway. **(G)** Densitometric analysis was performed to determine the relative ratios of each protein. Lamin A and β -actin were used as nuclear and cytoplasmic markers, respectively. The results are representative of three independent experiments and expressed as mean \pm SD. ##P < 0.01 compared with control group; **P < 0.01 compared with conditioned media group. **(H)** The transcriptional activities of p-NF- κ B p65 in HCT116 and HT29 cells in all groups were determined by Luciferase activity assay. The results are representative of three independent experiments and expressed as mean \pm SD. ##P < 0.01 compared with control group; *P < 0.05, **P < 0.01 compared with conditioned media group. **(I–M)** HCT116 and HT29 cells were treated with IGF-1 (20 ng/ml) with or without wogonoside with for 24 h. **(I)** Ki67 cell proliferation detection of HCT-116 and HT29 cells treated with IGF-1 with or without wogonoside. **(J)** The protein expression of PI3K, p-Akt, Akt, p-IKK α , IKK α , p-I κ B α and I κ B α was analyzed by Western Blot. **(K)** Densitometric analysis to determine the relative ratio normalized to β -actin. The results are representative of three independent experiments and expressed as mean \pm SD, ## P<0.01 compared with control group; **P<0.01 compared with IGF-1 group. **(L)** NF- κ B p65 nuclear translocation in HCT116 and HT29 cells in all groups were determined by Western Blot. **(M)** Densitometric analysis was performed to determine the relative ratios of each protein. Lamin A and β -actin were used as nuclear and cytoplasmic markers, respectively. The results are representative of three independent experiments and expressed as mean \pm SD. ##P < 0.01 compared with control group; **P < 0.01 with IGF-1 group. **(N)** The transcriptional activities of p-NF- κ B p65 induced by IGF-1 in HCT116 and HT29 cells were determined by Luciferase activity assay. The results are representative of three independent experiments and expressed as mean \pm SD. ##P < 0.01 compared with control group; **P < 0.01 with IGF-1 group.