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Erratum

The following was originally published in Volume 26, No. 2, pages 209–217, 2018 (doi: https://doi.org/10.3727/096504017X14944585873622). In the original article there were inadvertent mistakes for images of Western blot in Figures 2, 3, 4, 5, and 6. The figures have been reorganized using images from repeated experiments to rectify the errors. Corrected versions of the figures are shown here and the figures have been replaced with the corrected versions in the original published article in the online site (https://www.ingentaconnect.com/contentone/cog/or/2018/00000026/00000002/art00005). The corrections do not change any conclusion of the article. The authors apologize for any inconvenience caused.

Procaine Inhibits the Proliferation and Migration of Colon Cancer Cells Through Inactivation of the ERK/MAPK/FAK Pathways by Regulation of RhoA

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Colon cancer is one of the most lethal varieties of cancer. Chemotherapy remains as one of the principal treatment approaches for colon cancer. The anticancer activity of procaine (PCA), which is a local anesthetic drug, has been explored in different studies. In our study, we aimed to explore the anticancer effect of PCA on colon cancer and its underlying mechanism. The results showed that PCA significantly inhibited cell viability, increased the percentage of apoptotic cells, and decreased the expression level of RhoA in HCT116 cells in a dose-dependent manner ($p < 0.05$ or $p < 0.01$). Moreover, PCA increased the proportion of HCT116 cells in the G_1 phase as well as downregulated cyclin D1 and cyclin E expressions ($p < 0.05$). In addition, we found that PCA remarkably inhibited cell migration in HCT116 cells ($p < 0.01$). However, all these effects of PCA on cell proliferation, apoptosis, and migration were significantly reversed by PCA + pc-RhoA ($p < 0.05$ or $p < 0.01$). PCA also significantly decreased the levels of p-ERK, p-p38MAPK, and p-FAK, but PCA + pc-RhoA rescued these effects. Furthermore, the ERK inhibitor (PD098059), p38MAPK inhibitor (SB203580), and FAK inhibitor (Y15) reversed these results. These data indicate that PCA inhibited cell proliferation and migration but promoted apoptosis as well as inactivated the ERK/MAPK/FAK pathways by regulation of RhoA in HCT116 cells.

Key words: Colon cancer; HCT116; Procaine (PCA); RhoA; ERK/MAPK/FAK

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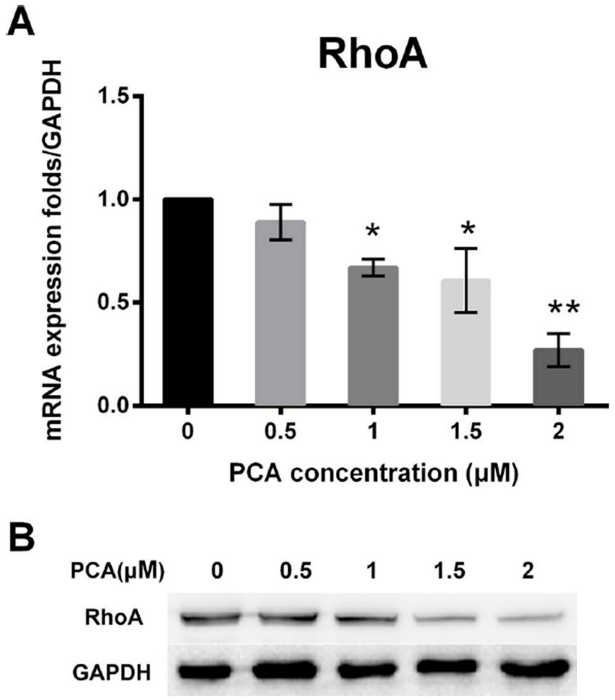

Figure 2. PCA inhibited the expression of RhoA in HCT116 cells. (A) PCA inhibited the mRNA expression level of RhoA at the concentrations of 1, 1.5, and 2 μM. (B) PCA inhibited the protein expression level of RhoA at the concentrations of 1, 1.5, and 2 μM. $*p < 0.05$, $**p < 0.01$.

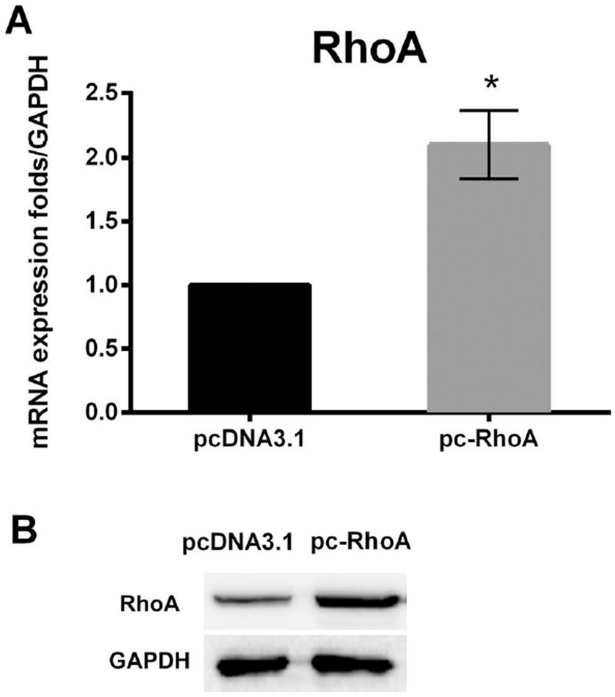

Figure 3. RhoA overexpression upregulated RhoA expression in HCT116 cells. (A) The level of RhoA mRNA was significantly upregulated by pc-RhoA. (B) The level of RhoA protein was significantly upregulated by pc-RhoA. $*p < 0.05$.

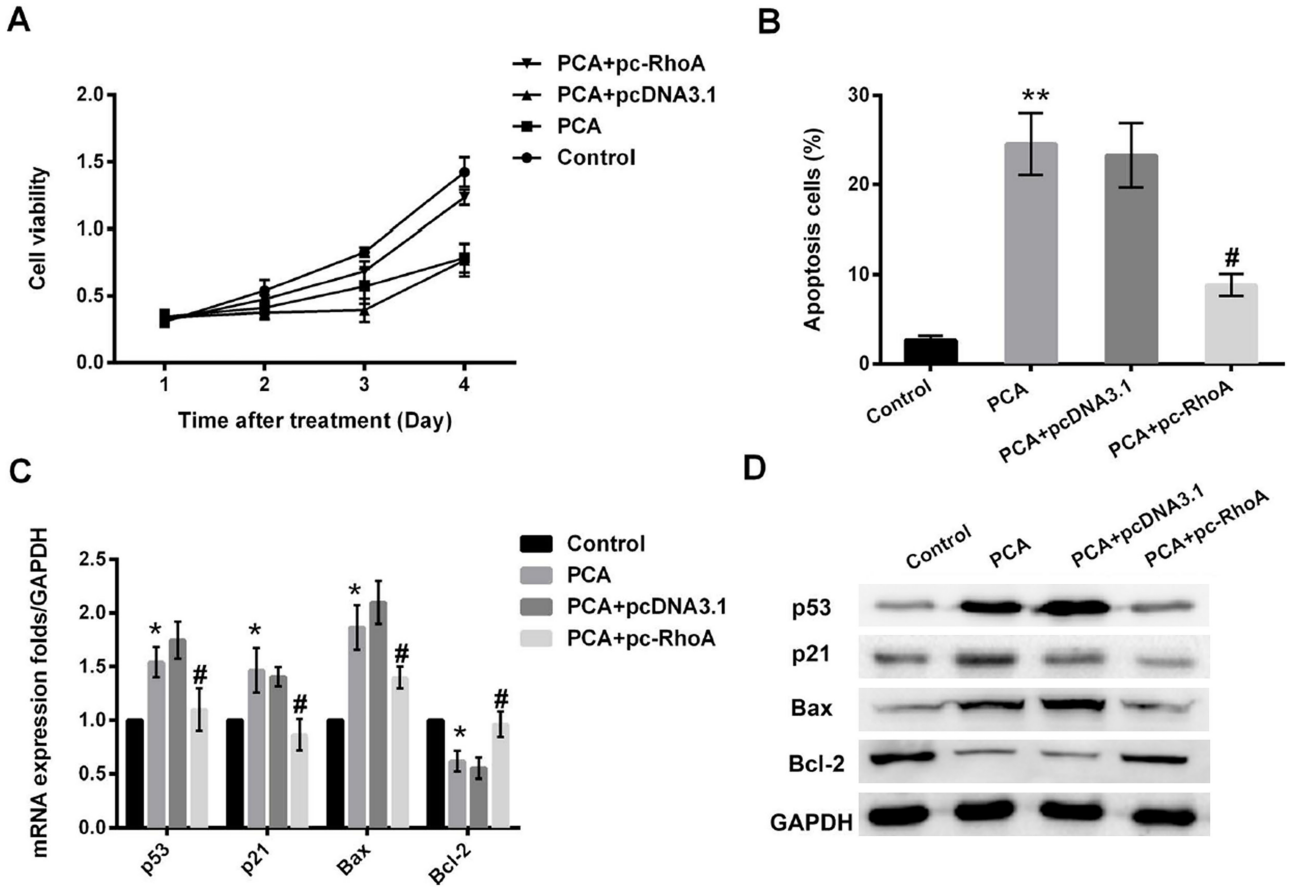

Figure 4. PCA inhibited cell viability and promoted apoptosis by regulating RhoA. (A) PCA + pc-RhoA remarkably promoted cell viability compared to PCA + pcDNA3.1. (B) PCA + pc-RhoA remarkably inhibited cell apoptosis compared to PCA + pcDNA3.1. (C) PCA + pc-RhoA downregulated p53, p21, and Bax mRNA expressions and upregulated Bcl-2 mRNA expression compared to PCA + pcDNA3.1. (D) PCA + pc-RhoA downregulated p53, p21, and Bax protein expressions and upregulated Bcl-2 protein expression compared to PCA + pcDNA3.1. *$p < 0.05$, **$p < 0.01$ for PCA compared with the control; #$p < 0.05$ for PCA + pc-RhoA compared with PCA + pcDNA3.1.

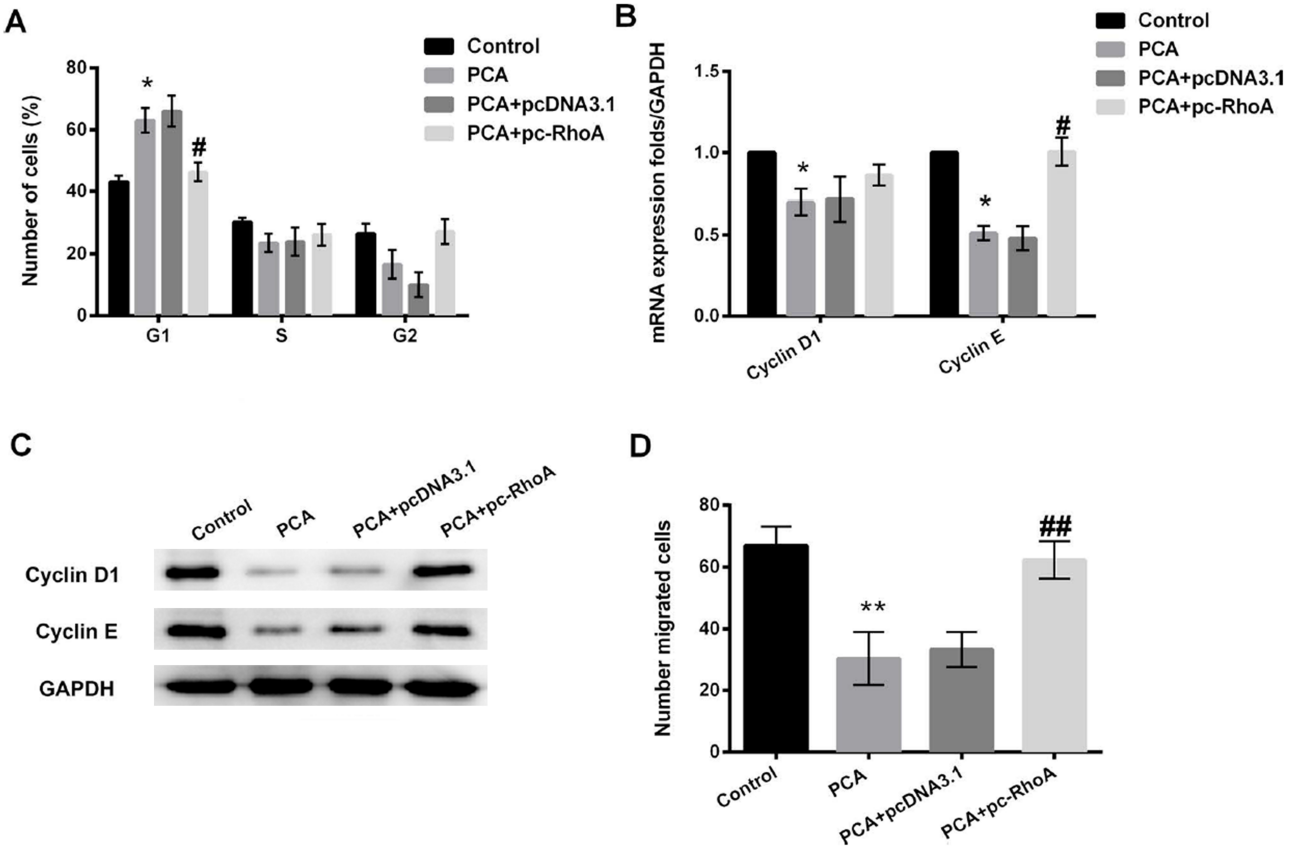

Figure 5. PCA arrested the cell cycle at the G_1 stage and suppressed cell migration by regulating RhoA. (A) PCA increased the proportion of HCT116 cells in the G_1 phase when compared with the control. (B) PCA downregulated the expressions of cyclin D1 and cyclin E mRNAs. (C) PCA reduced the expressions of cyclin D1 and cyclin E proteins. (D) PCA remarkably inhibited cell migration in HCT116 cells compared to the control. $*p < 0.05$, $**p < 0.01$ for PCA compared to control; $\#p < 0.05$ for PCA + pc-RhoA compared with PCA + pcDNA3.1; $\#\#p < 0.01$ for PCA + pc-RhoA compared with PCA + pcDNA3.1.

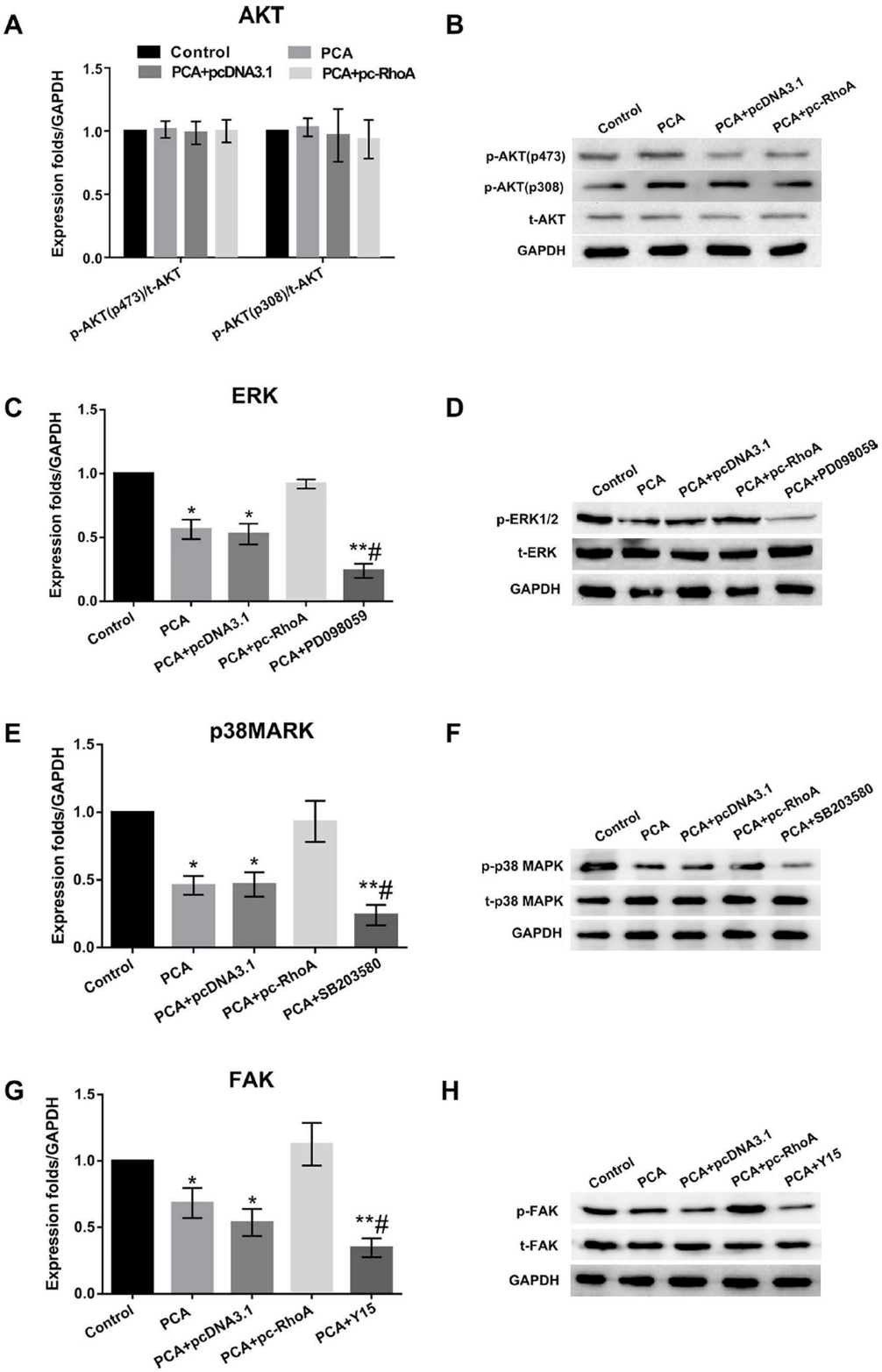

Figure 6. PCA inactivated the ERK/MAPK/FAK signaling pathways by regulating RhoA. (A) PCA had no effect on the AKT pathway. (B) PCA had no effect on the expression of AKT pathway-associated proteins. (C–H) mRNA and protein levels of the ERK p38MAPK and FAK pathway-associated factors were increased by PCA + pc-RhoA, but the activation effect was reversed by inhibitors of ERK1/2 (PD098059), p38MAPK (SB203580), and FAK (Y15). *$p < 0.05$, **$p < 0.01$ when compared with the control group, #$p < 0.05$ when compared with the PCA group.