Correction to "Protease-Activated Receptors Differentially Regulate Human Platelet Activation through a Phosphatidic Acid-Dependent Pathway"

In the above article [Holinstat M, Voss B, Bilodeau ML, and Hamm HE (2007) *Mol Pharmacol* **71:**686–694], parts of Figs. 3A and 5A were duplicated. The experiments for Figs. 3 and 5 were repeated, and the new results confirm those originally reported. The new experiments and results do not change the conclusions of the article. The corrected figures and their legends appear below.

The authors regret this error and apologize for any confusion or inconvenience it may have caused.

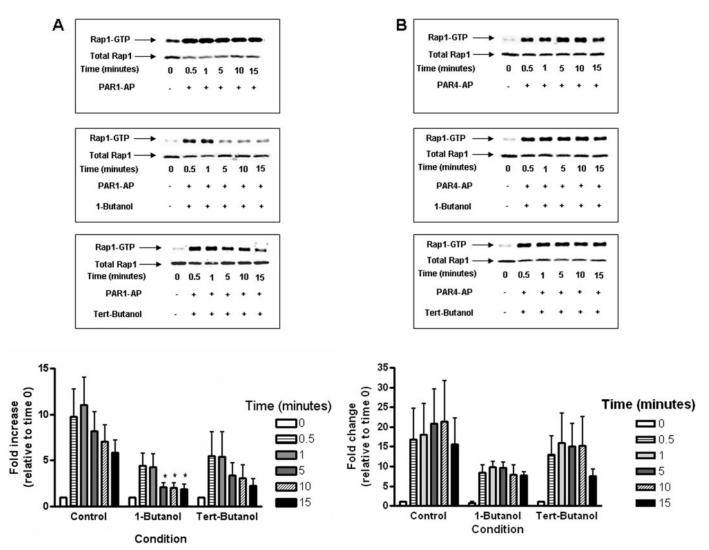


Fig. 3. Propranolol regulation of Rap1 activity. A, washed platelets were pretreated with or without 100 μ M propranolol followed by stimulation for various times with 20 μ M PAR1-AP. Active Rap1 (Rap1-GTP) was measured using a Rap1 activation assay (n=6). B, change in PAR1-AP-mediated Rap1 activation after propranolol treatment. Statistical analysis was based on a comparison of Rap1 activation at 0.5 min relative to other time points for Rap1 activation in a given treatment group. (P values for 1, 5, 10, and 15 min in the control condition were 0.1155, 0.2410, 0.0907, and 0.0556, respectively. P values for 1, 5, 10, and 15 min in propranolol treated conditions were 0.6173, 0.0031, 0.0036, and 0.0069, respectively (**, P < 0.01; n = 6). C, washed platelets were pretreated with or without 100 μ M propranolol followed by stimulation for various times with 200 μ M PAR4-AP (n = 3). D, change in PAR4-AP-mediated Rap1 activation after propranolol treatment. Statistical analysis was based on a comparison of Rap1 activation at 0.5 min relative to other time points for Rap1 activation in a given treatment group. (P values for 1, 5, 10, and 15 min in the control condition were 0.0837, 0.1853, 0.2344, and 0.4603, respectively. P values for 1, 5, 10, and 15 min in propranolol treated conditions were 0.3479, 0.1833, 0.3848, and 0.9799, respectively.

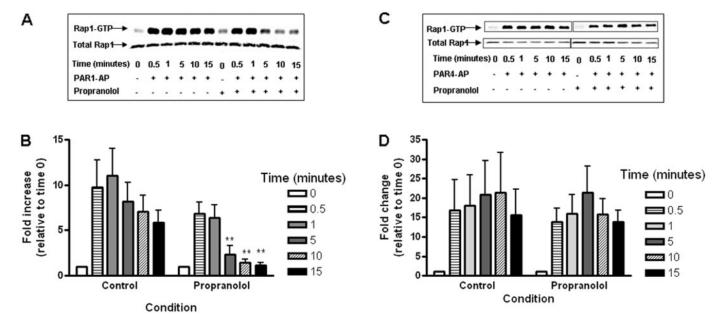


Fig. 5. 1-Butanol attenuates Rap1 activation. Washed platelets were pretreated with or without 0.4% 1-butanol or 0.4% tert-butanol for 4 min. A, after pretreatment, platelets were stimulated with $20~\mu\text{M}$ PAR1-AP for various times (0-15~min). Top indicates Rap1 activation without pretreatment (n=8), middle shows Rap1 activation after pretreatment with 1-butanol (n=6), and bottom shows Rap1 activation after pretreatment with tert-butanol (n=5). Active Rap1 (Rap1-GTP) was measured using a Rap1 activation assay. Bar graph indicates the -fold change in Rap1 activation compared with the unstimulated condition (average \pm S.E.M.). Statistical analysis was based on a comparison of Rap1 activation at 0.5 min relative to other time points for Rap1 activation in a given treatment group. (P values for 1, 5, 10, and 15 min in the control condition were 0.1155, 0.2410, 0.0907, and 0.0556, respectively. P values for 1, 5, 10, and 15 min in tert-butanol-treated conditions were 0.7387, 0.1319, and 0.1555, respectively. B, after pretreatment, platelets were stimulated with $200~\mu\text{M}$ PAR4-AP for various times (0-15~min). Top, Rap1 activation without pretreatment; middle, Rap1 activation after pretreatment with 1-butanol; bottom, Rap1 activation after pretreatment with tert-butanol (n=3). Active Rap1 (Rap1-GTP) was measured using a Rap1 activation assay. Bar graph indicates the -fold change in Rap1 activation compared with the unstimulated condition (average \pm S.E.M.). Statistical analysis was based on a comparison of Rap1 activation at 0.5 min relative to other time points for Rap1 activation in a given treatment group. (P values for 1, 5, 10, and 15 min in the control condition were 0.0837, 0.1853, 0.2344, and 0.4603, respectively. P values for 1, 5, 10, and 15 min in tert-butanol-treated conditions were 0.2225, 0.2036, 0.5003, and 0.5103, respectively. P values for 1, 5, 10, and 15 min in tert-butanol-treated conditions were 0.4391, 0.3194, 0.5575, and 0.3856, respectively (*, P < 0.05; n = 3)