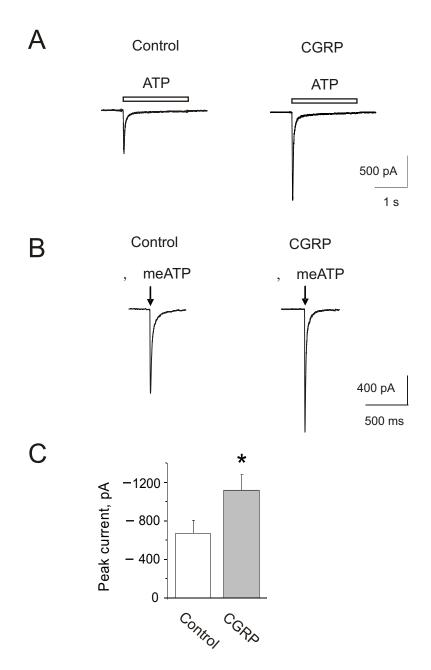


Supplemental Figure 1. CGRP binding to TG neurons. *A*, microphotograph depicts TG neurons labeled with rhodamine conjugated CGRP (0.5 M; red) and anti-tubulinIII antibody (green). Calibration bar = 50 m. *B*, Somatic size distribution of TG neurons immunostained with anti-tubulinIII antibody (filled columns) and labeled with rhodamine-conjugated CGRP (dashed bars). Data are from approximately 1,500 cells (3 independent experiments). *C*, CGRP (1 μ M, 1 h) has no effect on the amplitude of currents elicited by application of capsaicin (1 μ M, 2 s). *D*, Microphotograph depicts TG neurons labeled with rhodamine conjugated CGRP (0.5 M; red) and anti-TRPV1 antibody (green). Calibration bar = 50 m.



Supplemental Figure 2. CGRP treatment (1 μ M, 1 h) induces potentiation of peak currents elicited by ATP or , meATP. *A*, Examples of currents evoked in TG neurons by application of ATP (10 μ M, 2 s) in control condition (left) or after CGRP treatment (right). The mean peak current amplitude induced by ATP after 1 h CGRP exposure is 180 ± 38 % of control (n = 23), indicating analogous potentiation as observed with , -meATP. *B*, CGRP (1 μ M, 1 h) increases currents elicited by short (10 ms) pressure application of , -meATP (200 μ M). Examples of , meATP-evoked currents in control condition (left) or after 1 h CGRP treatment (right). This result shows that the CGRP-evoked enhancement of P2X₃ receptor function is not biased by the method of agonist application. *C*, Peak current amplitude evoked by puffer-applied , meATP is significantly increased by CGRP treatment (p = 0.044; n = 11) with respect to control (n = 10). Note that current deactivation is not affected by CGRP treatment because the rapid phase of current monoexponential decay was 23 ± 7 and 21 ± 5 ms for control and treated neurons (n = 9 and 8, respectively).