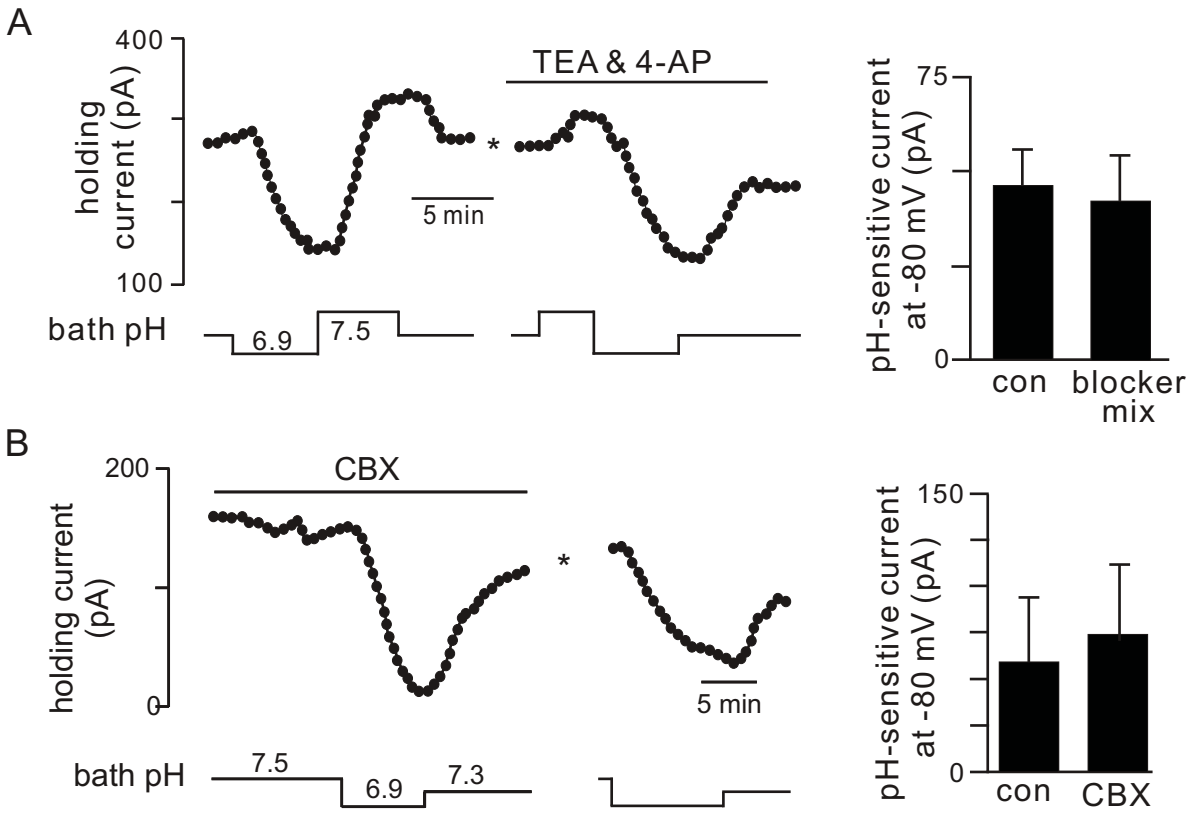
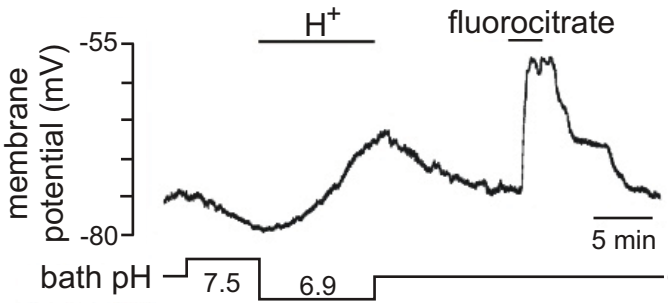


Supplemental Figure 1



Supplemental Figure 2

Supplemental Figure 1. The pH-sensitive current in RTN astrocytes in not blocked by TEA, 4-AP or CBX. A, traces of holding current and bath pH show the characteristic response of RTN astrocyte to changes in bath pH; acidification to 6.9 decreases holding current and alkalization to 7.5 increases holding current. At a control pH of 7.3, exposure to TEA (10 mM) and 4-AP (50 μM) had little effect on either holding current or pH-sensitivity. The bar graph on the right summarizes amplitude of the pH-sensitive current (at a holding potential of -80 mV) under control conditions and in the presence of TEA and 4-AP (blocker mix). The blocker mix had no effect on pH-sensitivity of RTN astrocytes. **B**, trace of holding current and bath pH show pH-sensitivity was retained when gap junctions were blocked with carbenoxalone (100 μM, CBX). In fact, we noted a modest increase in amplitude of the pH-sensitive current probably due to CBX-induced increase in membrane resistance. These results rule out possible contributions of most voltage-gated and Ca^{2+} dependent K^+ channels and possible pH sensors as well as indicate that pH-sensitivity is not conferred gap junctional currents. In panels A and B, the asterisks designate 10 minute time breaks.

Supplemental Figure 2. Fluorocitrate activates pH-sensitive RTN astrocytes. Trace of membrane potential from a pH-sensitive astrocyte perfused with HEPES buffer shows that increasing bath pH from 7.3 to 7.5 hyperpolarized membrane potential from -75 to -80 mV, whereas acidification to pH 6.9 depolarized membrane potential from -80 mV to -67 mV. At pH 7.3 exposure to fluorocitrate (100 μM) reversibly depolarized membrane potential ~ 17 mV. It should also be recognized that fluorocitrate is a poor H^+ mimetic and that it may affects other glial cell types in addition to pH-sensitive astrocytes.