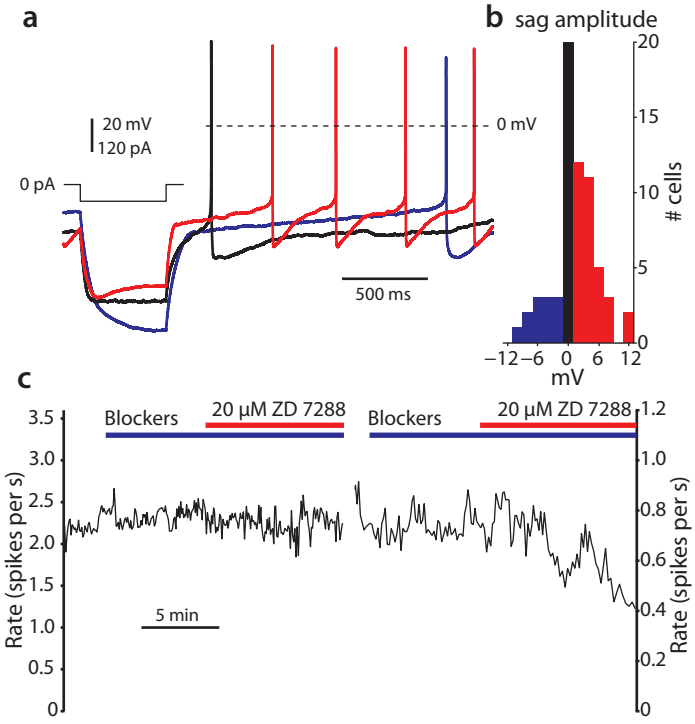
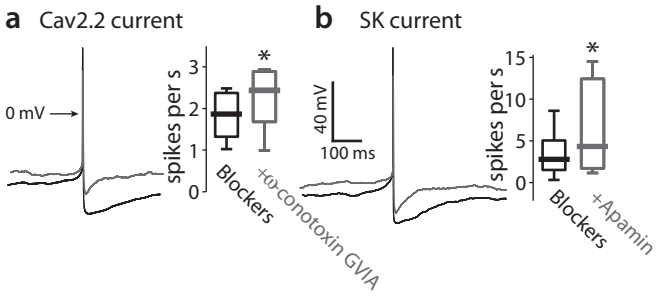
Calcium entry induces mitochondrial oxidant stress in vagal neurons at risk in Parkinson's disease

Joshua A. Goldberg, Jaime N. Guzman, Chad M. Estep, Ema Ilijic, Jyothisri Kondapalli, Javier Sanchez-Padilla and D. James Surmeier



Supplementary Figure 1 HCN currents in cholinergic DMV neurons. (**a**) DMV neurons were heterogeneous with regard to expression of HCN currents, which activate in response to a 60 pA hyperpolarizing pulse causing the voltage to depolarize (sag). (**b**) distribution of the size of the voltage sag reveals that approximately half of the cells (33/65) exhibit a sag (shown in red). (**c**) autonomous firing rate measured in cell attached configuration was not affected by washing on of 20 µM ZD 7288 in 4/7 cells (left panel) but slowed down pace-making in the remaining 3 (right panel).



Supplementary Figure 2 Contribution of Cav2 and SK channels to discharge patterns in DMV neurons. (**a**) application of 1 μM ω-conotoxin GVIA, a Cav2.2 (N-type) calcium channel blocker, reduced the mAHP that followed each spike during pacemaking, resulting in a mild but significant speeding up of the firing rate [median increase of 28% in firing rate, $n = 7$, $P < 0.05$, Wilcoxon signed-rank test (SRT) for paired samples]. (**b**) the SK channel blocker, apamin, affected the mAHP in a similar fashion and resulted in a significant and larger increase in firing rate (median increase of 61% in firing rate, $n = 6$, $P < 0.05$, SRT).