rTg4510 tau mutant mice (TG) exhibit progressive development of neurofibrillary tangles, neuronal death, and cognitive impairment. In Rocher et al. (2010) we established that there is striking dendritic regression and increased excitability of cortical pyramidal cells from these mice at 8.5 months-of-age (M). The present study assessed the effects of progressive abnormal tau expression on structural and functional properties of cells from 1, 3, 8.5 and 13M TG and wild-type (WT) mice. Layer 3 frontal cortical pyramidal cells in \textit{in vitro} slices from TG and WT mice were characterized using whole-cell patch-clamp recordings. Cells were simultaneously filled with biocytin then subsequently imaged and reconstructed in 3D. TG cells were more intrinsically excitable than WT at every age, exhibiting depolarized resting potentials, increased sag potentials and increased action potential firing rates. At 3M the frequency of spontaneous excitatory postsynaptic currents was decreased, whereas at the other 3 ages frequency was either the same or increased in TG versus WT cells. TG cells did not differ from WT with regard to dendritic parameters at 1 and 3M, but by 8.5M they exhibited significantly reduced dendritic arbors and spines, but increased axonal boutons, and by 13M they were severely dystrophic. The preservation of excitatory synaptic responses in cells that have undergone significant dendritic atrophy and spine loss demonstrates that compensatory homeostatic synaptic scaling occurs during progressive tauopathy in these mice. Increased intrinsic excitability and axonal bouton density are possible contributors to maintained synaptic homeostasis in the face of major structural changes.