Kinesins, including the kinesin 2/KIF3 molecular motor, play an important role in intracellular traffic and can deliver vesicles to distal axon terminal, to cilia, to non-polarized cell surface or to epithelial cell basolateral membrane, thus taking part to the establishment of cellular polarity. We report here the consequences of the kinesin 2 motor inactivation in the thyroid of 3 week-old $Kif3a^{\Delta/flox}$ $Pax8^{Cre/+}$ mutant mice. Our results indicate first that 3 week-old $Pax8^{Cre/+}$ mice used in these experiments present minor thyroid functional defects resulting in a slight increase in circulating bioactive TSH and intracellular cAMP levels, sufficient to maintain blood T4 levels in the normal range. Second, Kif3a inactivation in thyrocytes markedly amplified the phenotype observed in $Pax8^{Cre/+}$ mice, resulting in an altered TSH signaling upstream of the second messenger cAMP and mild hypothyroidism. Finally, our results in mouse embryonic fibroblasts indicate that Kif3a inactivation in the absence of any Pax8 gene alteration leads to altered GPCR plasma membrane expression, as shown for the β2 adrenergic receptor, and we suggest that a similar mechanism may explain the altered TSH signaling and mild hypothyroidism detected in $Kif3a^{\Delta/flox}$ $Pax8^{Cre/+}$ mutant mice.