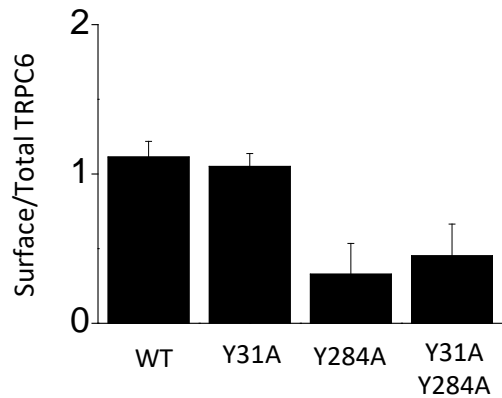
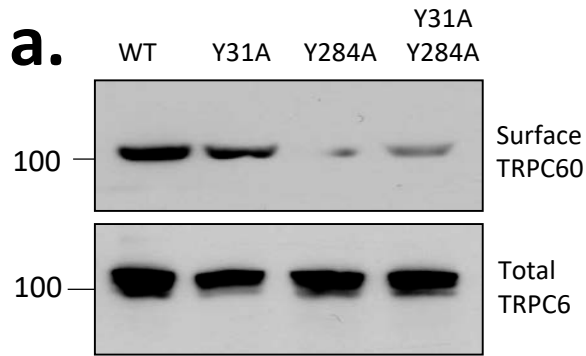


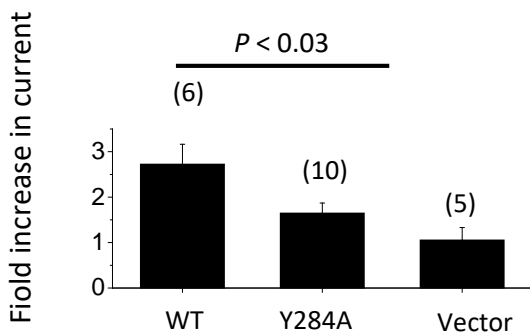
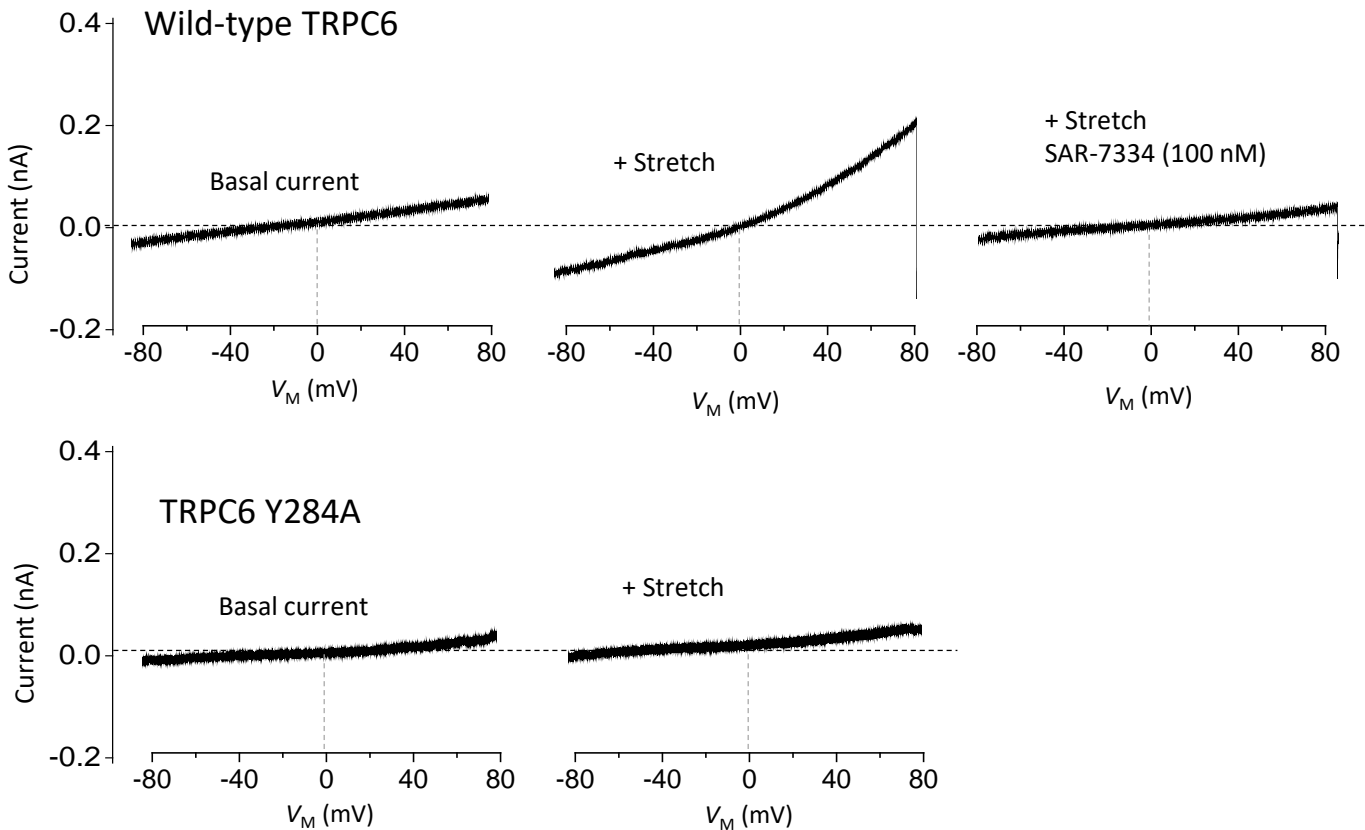
## **Supplemental Data**

### **Mechanisms underlying modulation of podocyte TRPC6 channels by suPAR: Role of NADPH oxidases and Src family tyrosine kinases**

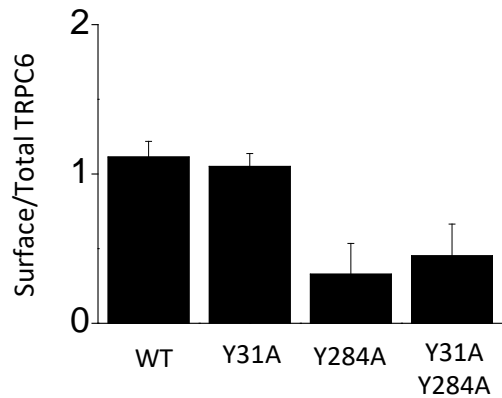
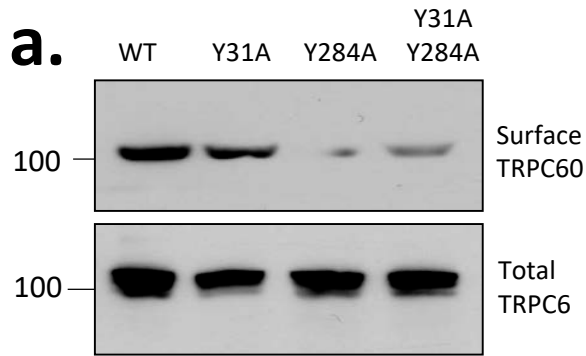
Eun Young Kim, Naghmeh Hassanzadeh  
Khayat, Stuart E. Dryer



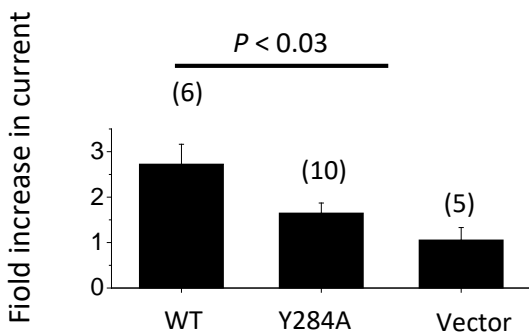
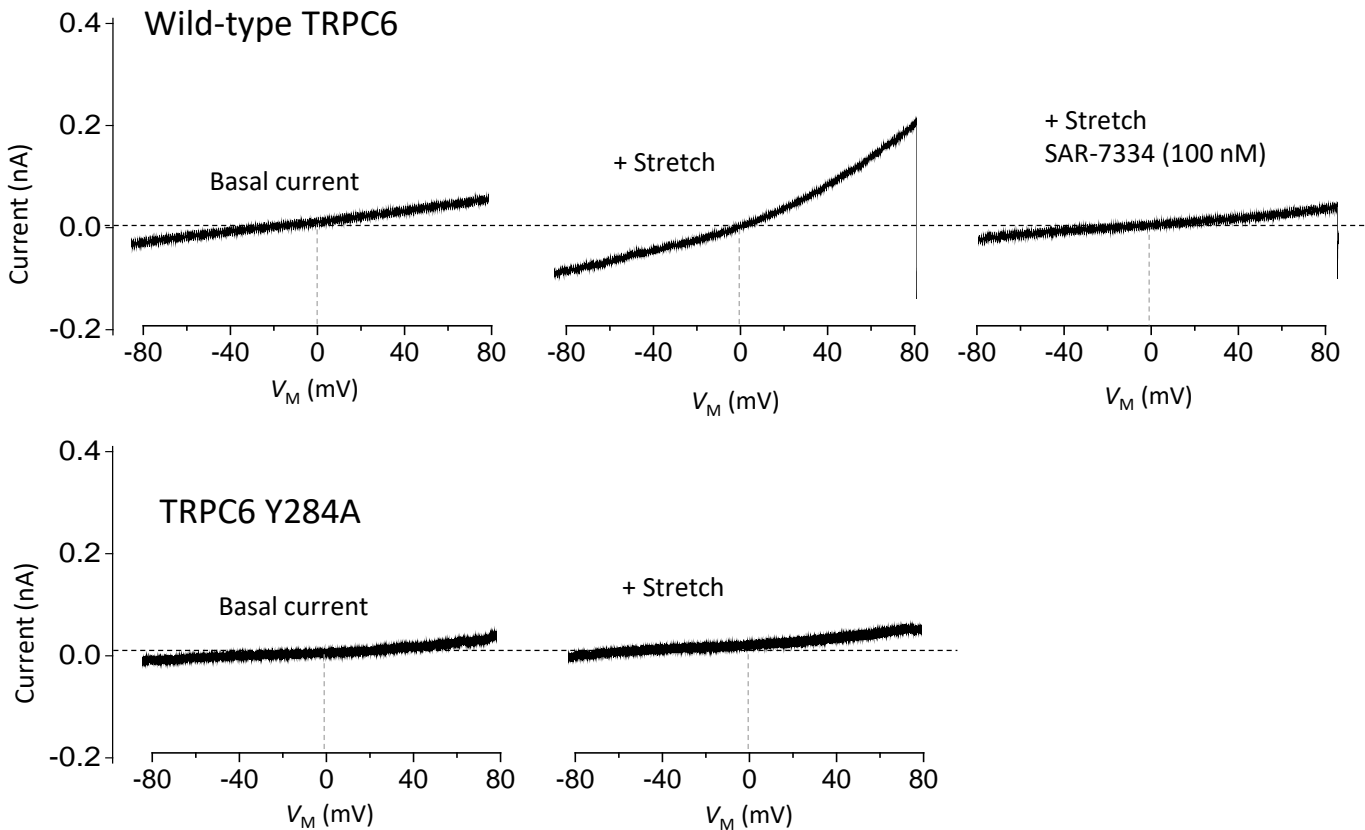
**b.**



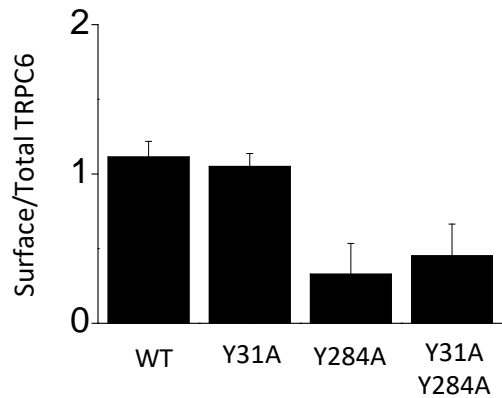
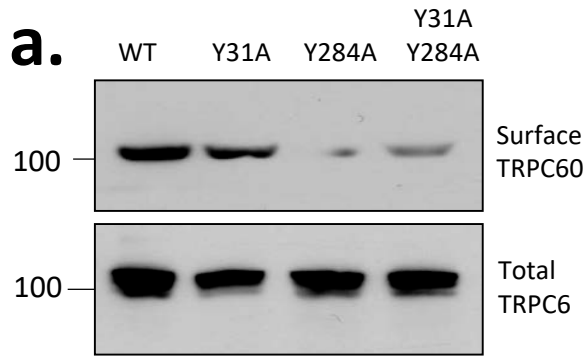
Supplement Figure 3. TRPC6 channels expressed in CHO-K1 cells showing effects of mutations of two tyrosines known to be phosphorylated by Src family kinases in glomeruli [41]. TRPC6 Y284A does not exhibit normal trafficking to the cell surface (a) and is a functional hypomorph (b), as it does not exhibit normal activation in response to membrane stretch in whole-cell recordings. An essentially identical effect was previously reported with a Y284F mutation expressed in HEK-293 cells [41].



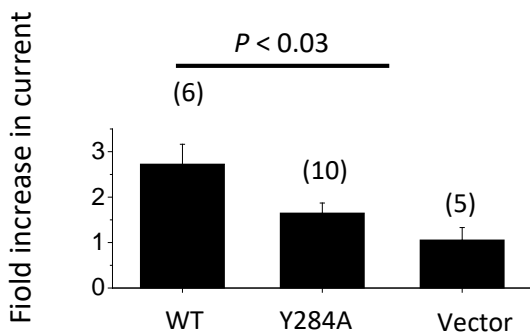
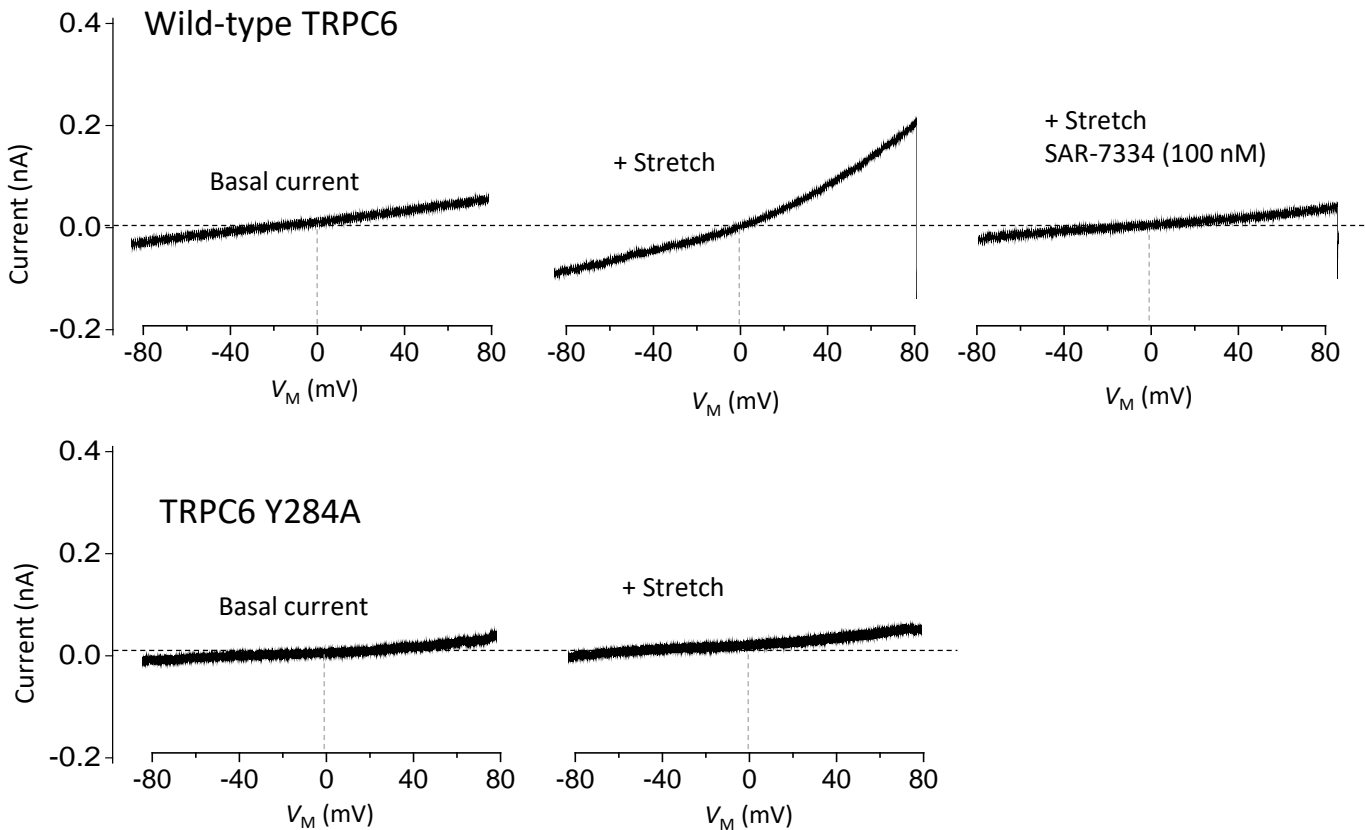
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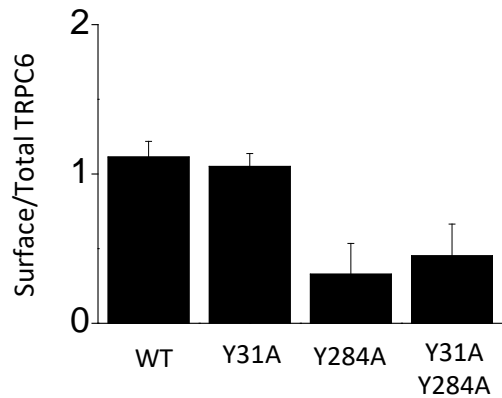
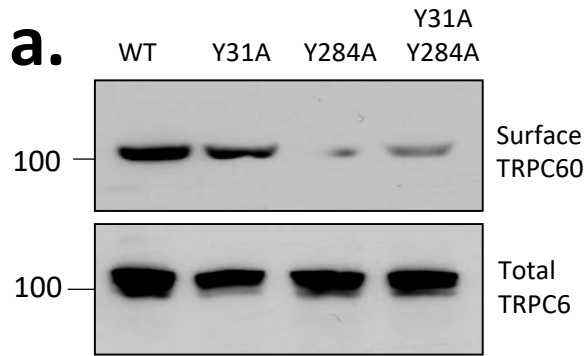
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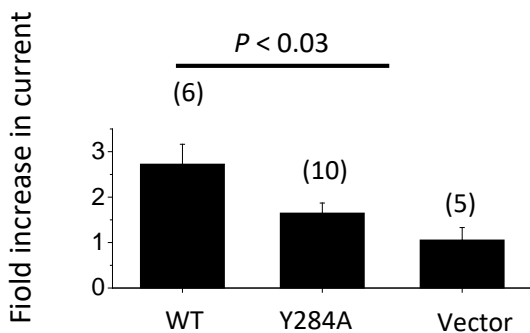
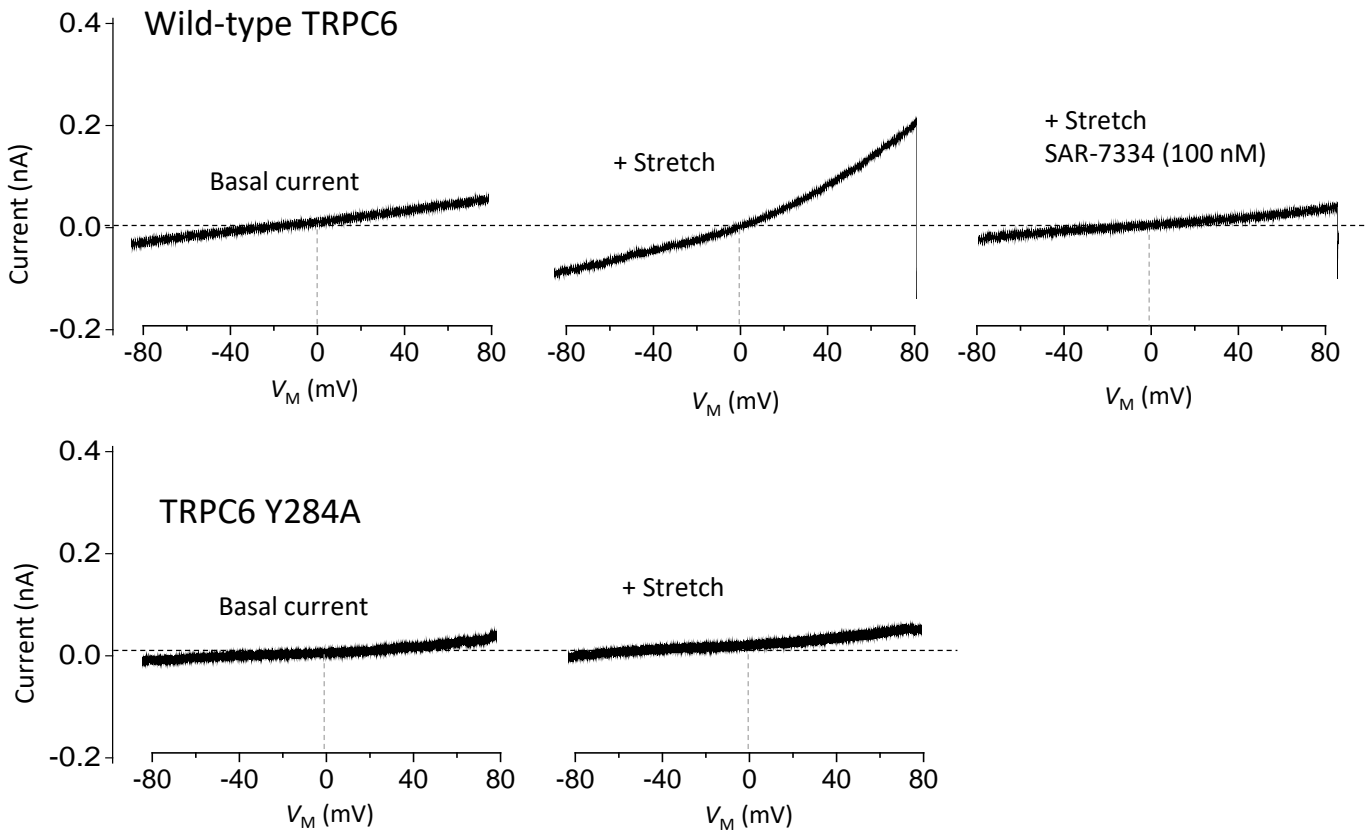
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**b.**



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