

Endoplasmic Reticulum-Associated Degradation of the Renal Potassium Channel, ROMK, Leads to Type II Bartter Syndrome

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Tables depicting Primers and Yeast Strains used in this study with corresponding references

Table S1: Oligonucleotide Primers

Primer	Use	Sequence
A198TF	A198T point mutation – forward	5'-cggtcagcaagaatacggtgatcagcaagc-3'
A198TR	A198T point mutation - reverse	5'-gcttgctgatcacgtattctgctgaacg-3'
R212PF	R212P point mutation - forward	5'-ctgcctccatcccagggtggccaatcttag-3'
R212PR	R212P point mutation - reverse	5'-ctaagattggccactggatgaggaggcag-3'
H270YF	H270Y point mutation - forward	5'-cccactgacgatctactacattttgaccacaac-3'
H270YR	H270Y point mutation - reverse	5'-gttgtggtaataatgttagatcgtagtggg-3'
Y314CF	Y314C point mutation - forward	5'-gtccgcacgtcatgcgtccagaggag-3'
Y314CR	Y314C point mutation - reverse	5'-ctcctctggacgcacgtgcggac-3'

Table S2: Yeast Strains

Strain	Relevant Genotype	Source
BY4742	<i>MATα his3Δ, leu2Δ, ura3Δ</i>	Invitrogen
<i>pdr5Δ</i>	<i>MATα, his3Δ, leu2Δ, ura3Δ, pdr5::KANMX</i>	Invitrogen
<i>SSA1</i>	<i>MATα, his3-11,15, leu2-3,112, ura3-52, trp1-Δ1, lys2, ssa2-1(LEU), ssa3-1(TRP1), ssa4-2(LYS2)</i>	Ref. [1]
<i>ssa1-45</i>	<i>MATα, his3-11,15, leu2-3,112, ura3-52, trp1-Δ1, lys2, ssa1-45, ssa2-1(LEU), ssa3-1(TRP1), ssa4-2(LYS2)</i>	Ref. [1]
<i>cdc48-2</i>	<i>MATα his3Δ, leu2Δ, ura3Δ, cdc48-2::KANMX</i> (Back-crossed 3X to BY4742)	Ref. [2]
<i>trk1Δ trk2Δ</i>	<i>MATα his3Δ, leu2Δ, ura3Δ, lyp1Δ, trk1::URA3, trk2::NATMX, can1::Ste2pr-HIS3</i>	Ref. [3]
<i>trk1Δ trk2Δvps23Δ</i>	<i>can1Δ::STE2pr-HIS3 lyp1Δ his3Δ1 leu2Δ0 ura3Δ0 met15Δ0 trk1Δ::URA trk2Δ::NAT vps23Δ::KANMX</i>	Ref. [3]

References

1. Becker, J., et al., *Functional interaction of cytosolic hsp70 and a DnaJ-related protein, Ydj1p, in protein translocation in vivo*. Mol Cell Biol, 1996. **16**(8): p. 4378-86.
2. Moir, D., et al., *Cold-sensitive cell-division-cycle mutants of yeast: isolation, properties, and pseudoreversion studies*. Genetics, 1982. **100**(4): p. 547-63.

3. Kolb, A.R., et al., *ESCRT regulates surface expression of the Kir2.1 potassium channel*. Mol Biol Cell, 2014. **25**(2): p. 276-89.