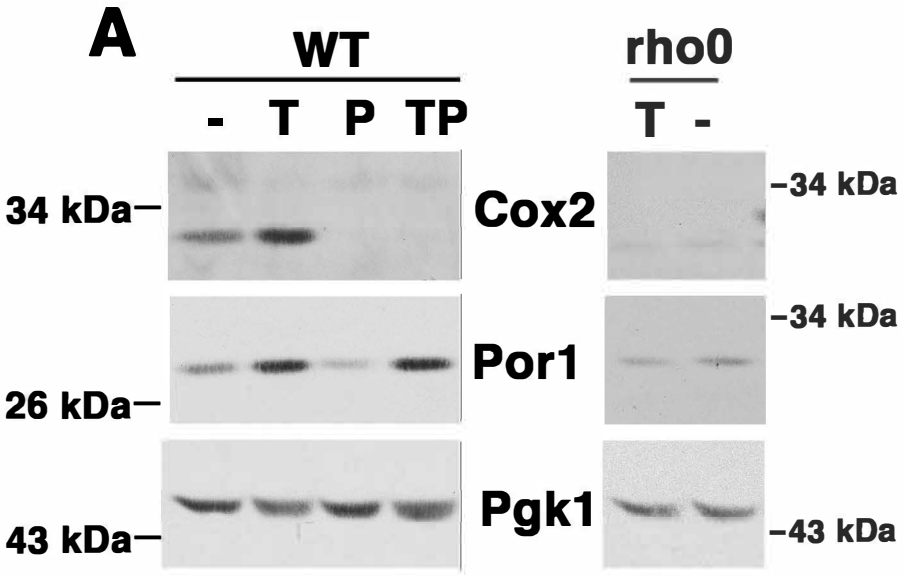
**Suppl. Fig. 1**



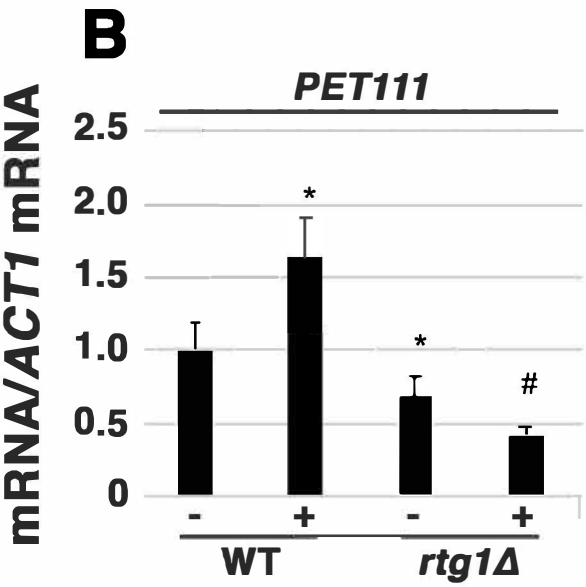



Fig. S1: (A) Cells were treated +/- tunicamycin (0.5 µg/ml) or pentamidine (100 µM) or both for 5h. Cell lysate was analyzed by Western blot for Cox2, Por1 and the loading control Pgk1. Pentamidine inhibited Cox2 but not Por1 protein expression as well as ER stress-mediated induction. In *rho0* cells, Por1 induction by tunicamycin was abrogated.

(B) mRNA was isolated from wild-type and *rtg1Δ* cells incubated +/- tunicamycin (0.5 µg/ml) for 5h. Error bars indicate SEM; $n \geq 5$ biological replicates. * indicates $p < 0.05$ compared to wild-type control. # indicates $p < 0.05$ compared to *rtg1Δ* control.

**Suppl. Fig. 2**

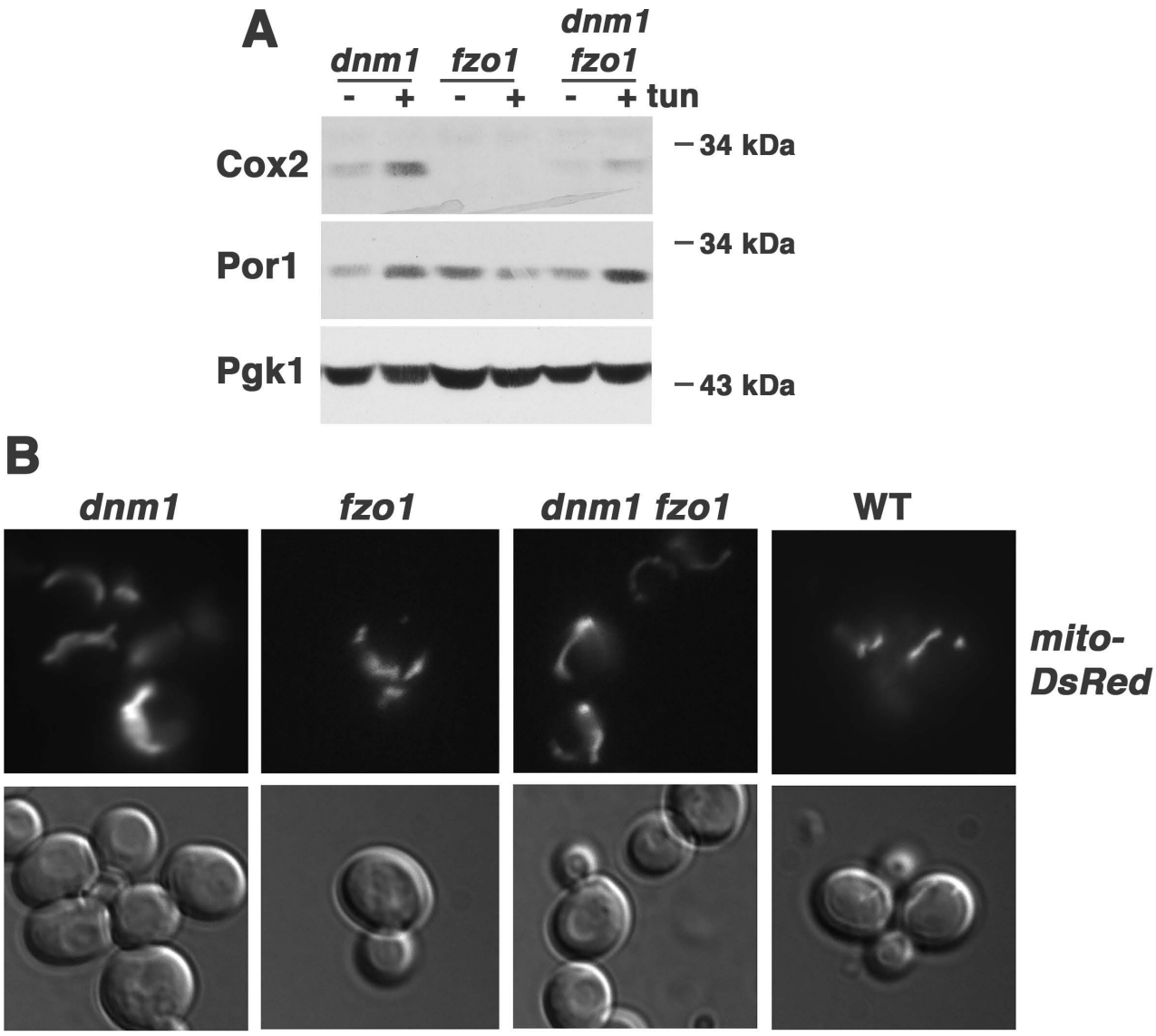



Fig. S2: ER stress induction of Cox2 and Por1 proteins in *dnm1Δ* and *fzo1Δ* cells. Cells were treated with tunicamycin (0.5 µf/ml) for 5h. (A) Lysate was analyzed by Western blot normalized to protein content. Pgk1 was detected as a loading control. (B) Cells bearing mito-dsRed were visualized by fluorescence microscopy to assess mitochondrial morphology; fragmented mitochondria were observed in *fzo1Δ* cells, elongated mitochondria were observed in *dnm1Δ* cells, and normal mitochondrial morphology was observed in wild-type and *fzo1Δ dnm1Δ* cells.

**Suppl. Fig. 3**

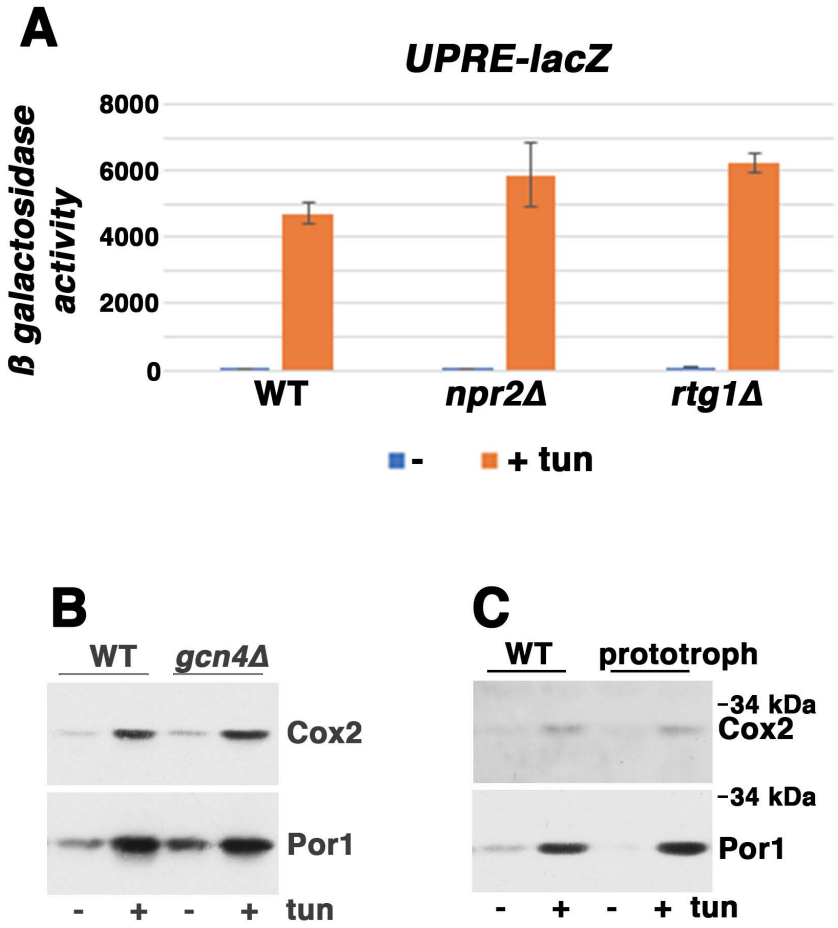



Fig. S3: (A) UPR induction in ER stressed cells. Exponentially growing cells bearing URA3-marked plasmids with UPRE-lacZ (pJC104) were treated with tunicamycin (0.5 µg/ml) for 5h. Cell lysate was prepared, ß galactosidase activity was assayed, and expressed as µmol/min/mg protein. Like wild-type cells, *rtg1Δ* and *npr2Δ* cells induced a UPR in response to tunicamycin. Error bars = SEM; n ≥ 3.

(B & C) Induction of mitochondrial proteins by ER stress is independent of amino acid deprivation. Western blots showing Cox2 and Por1 induction by ER stress. Exponentially growing cells were incubated with tunicamycin (0.5 µg/ml) for 5h. Lysates from wild-type and *gcn4Δ* cells (B), and wild-type cells and a prototroph (C) were analyzed by Western blot for Cox2 and Por1 proteins.

**Suppl. Fig. 4**

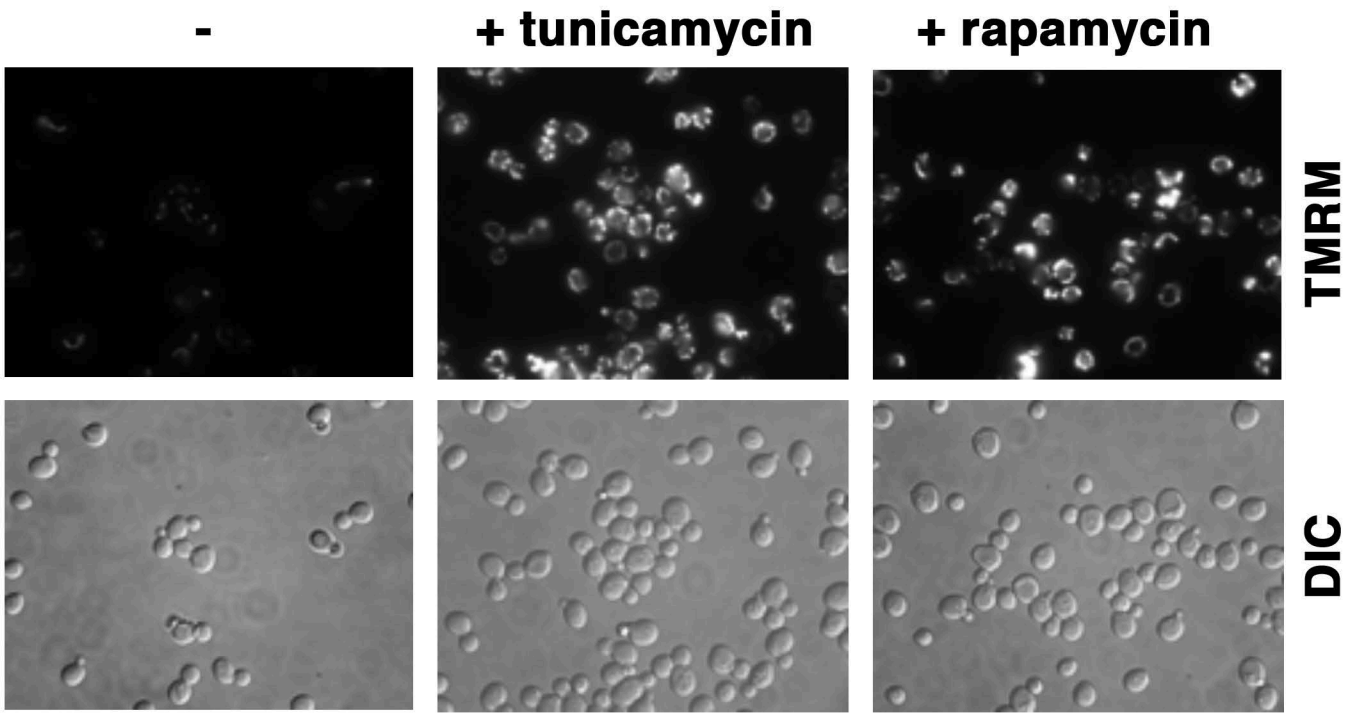



Fig. S4: Mitochondrial membrane potential staining upon inactivation of TORC1 signaling by rapamycin. Exponentially growing cells were treated or not with tunicamycin (0.5 µg/ml) or rapamycin (200 nM) for 5h. Cells were stained with TMRM (5 nM, nonquenching mode) for 30 min. and then visualized by fluorescence microscopy.

**Suppl. Fig. 5**

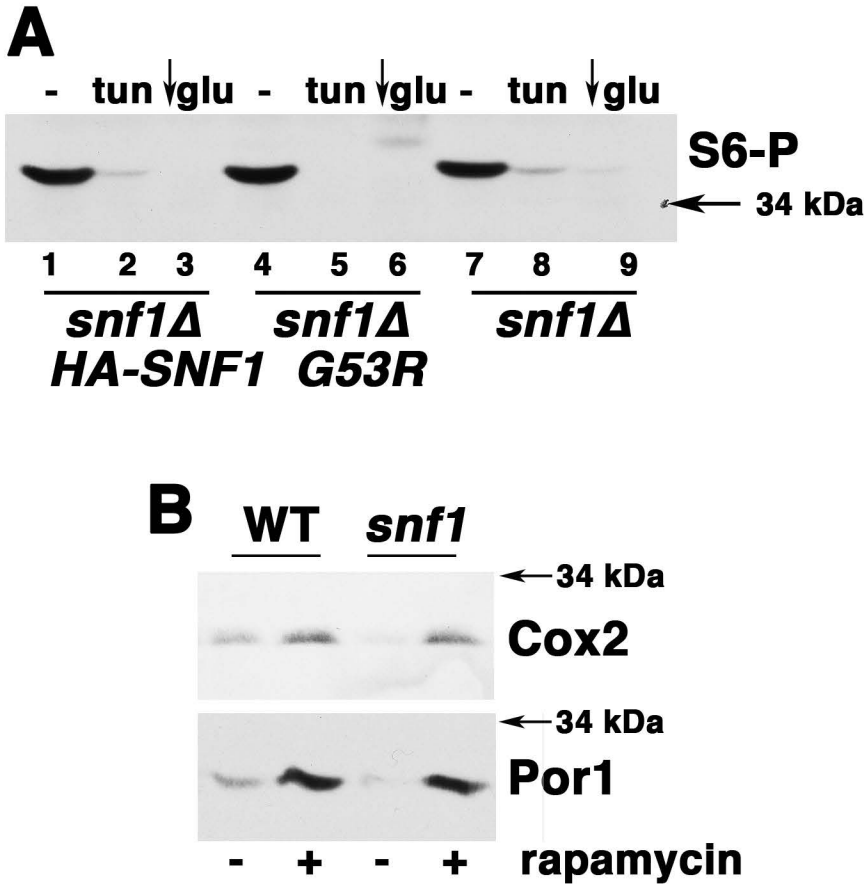



Fig. S5: Snf1 and TORC1 signaling in response to ER stress. (A) Western blot to detect Rps6 phosphorylation. Exponentially growing cells were treated with tunicamycin (0.5 µg/ml) for 5h. As a positive control, cells were shifted to 0.05% glucose for 1h (arrow). In cells overexpressing Snf1-G53R (lane 4, in the absence of tunicamycin), Rps6 phosphorylation was not affected, suggesting TORC activity is not repressed. In *snf1Δ* cells, tunicamycin-induced dephosphorylation of Rps6 was not impaired, suggesting TORC1 inactivation is not dependent on Snf1.

(B) Western blot showing Cox2 and Por1 levels in response to rapamycin. Exponentially growing cells were treated with or without 200 nM rapamycin for 5h. Induction of Cox2 and Por1 by rapamycin were similar in wild-type and *snf1Δ* cells, suggesting TORC1 inactivation is not dependent on Snf1.