Supplementary Figure 4. Msp1 and ATAD1 facilitate the turnover of Pex15 and Pex26, respectively. (Related to Figure 4)

(A) The indicated strains co-expressing GFP-Pex15 from the GAL1 promoter and RFP-SKL were cultured in galactose medium for 6 hr and imaged by fluorescence microscopy. (B) The wild-type and msp1Δ strain expressing GFP-Pex15 from the GAL1 promoter were grown in SGal medium for 5-6 hr to induce GFP-Pex15 accumulation and then pulsed in glucose medium. Whole-cell lysates were prepared from cells harvested every 30 min and analyzed by immunoblot. Por1 is the loading control. (C) The optical densitometry quantification of (B). The values represent mean ± SEM (n=3, * p<0.05, **<0.01, t-test, two-tail). (D) Representative images of the galactose promoter-based shut-off experiment shown in Figure 4E. Arrows mark the area of mitochondria. (E) The optical densitometry quantification of Figure 4E. The values represent mean ± SEM (n=3, * p<0.05, **<0.01, t-test, two-tail). (F) HeLa cells and HDFs stably expressing GFP-PEX26 protein were treated with the control siRNA (scr) or the siRNAs (#1-#3) against human ATAD1 for 6 days. Whole-cell lysates were harvested and immunoblotted with anti-ATAD1, GFP and QCR2 antibodies. QCR2 is a mitochondrial protein used as a loading control.