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CORRECTION

Correction: A ubiquitin–proteasome pathway degrades the inner nuclear membrane protein Bqt4 to maintain nuclear membrane homeostasis

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There were errors in *J. Cell Sci.* (2023) **136**, jcs260930 (doi:10.1242/jcs.260930).

The Cdc48 mutant used in the article was incorrectly reported as *cdc48-P267L/A550T*. This error arose due to differences in amino acid residue numbering of the Cdc48 sequence in the UniProt and PomBase databases, and as a result of incorrect design of a primer used for mutagenesis. The correct description of the Cdc48 mutant, based on the amino acid sequence of Cdc48 in PomBase (SPAC1565.08), is *cdc48-P251L/A544T*.

Fig. 5 has been updated to show the correct mutation sites in Fig. 5A and to refer to the Cdc48 mutant as *cdc48-P251L/A544T* throughout the figure and legend. The corrected and original figures are shown below.

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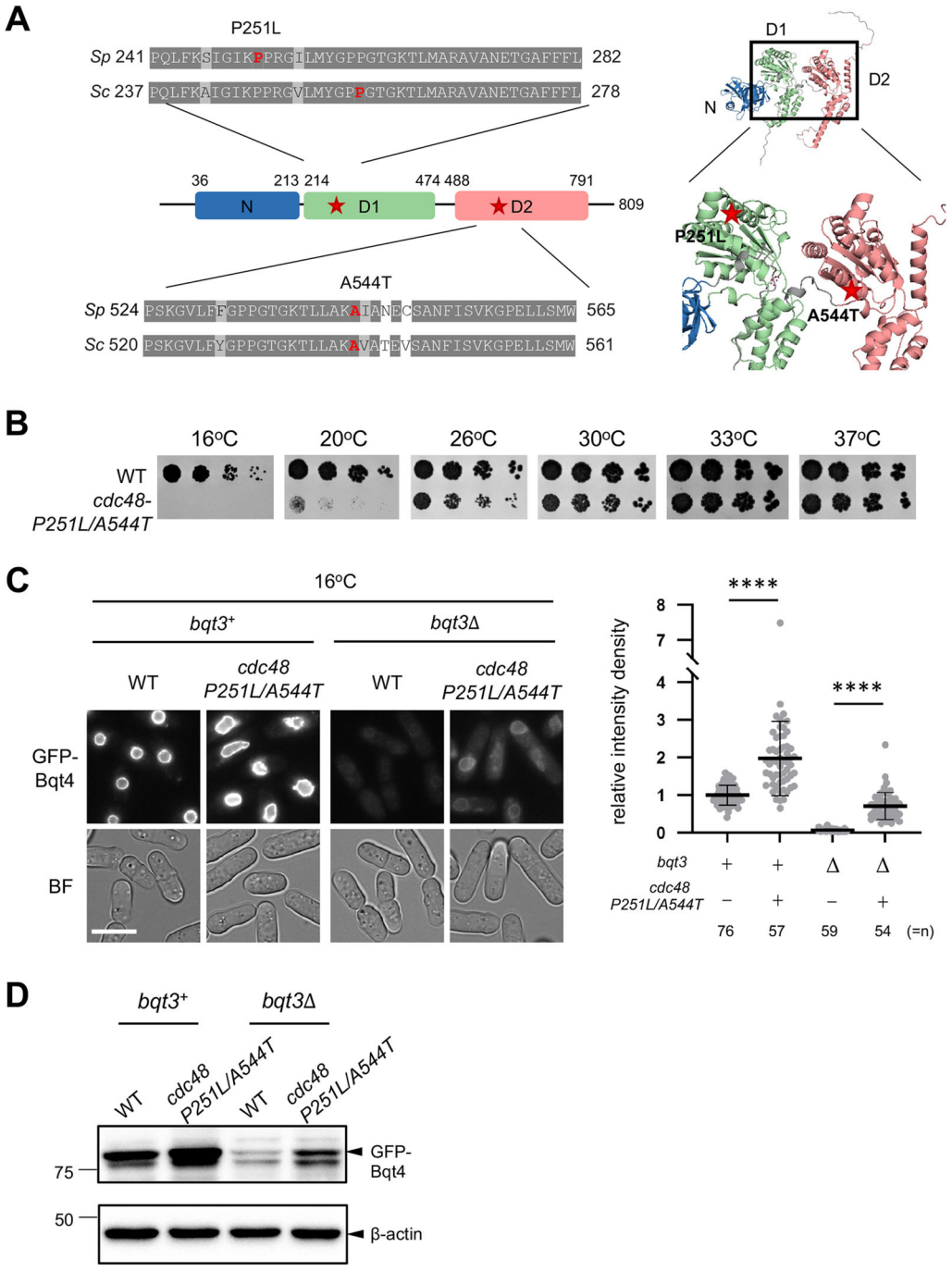

Fig. 5 (corrected figure). Removal of Bqt4 requires the Cdc48 ATPase complex. (A) The Cdc48 mutant used in this study. Left panel: amino acid sequence alignment around the D1 and D2 domains of *S. pombe* (*Sp*) and *S. cerevisiae* (*Sc*) Cdc48. Gray and light gray shades denote identical and similar amino acids, respectively (top and bottom). The amino acids indicated in red (P251 and A544; corresponding to red stars in the schematic diagram in the middle) are both mutated in this study (P251L/A544T). Right panel: the mutation sites are shown in the predicted three-dimensional structure of Cdc48 by AlphaFold2 (https://alphafold.ebi.ac.uk/entry/Q9P3A7). (B) Temperature sensitivity of *cdc48-P251L/A544T* mutant. Fivefold serially diluted cells harboring *cdc48-P251L/A544T* mutant were spotted on YES plates and cultured at different temperatures as indicated at the top. (C,D) Effect of hypomorphic mutation of *cdc48* on Bqt4 degradation. Cells harboring the *cdc48-P251L/A544T* mutant in the *bqt3*$^{+}$ or *bqt3*Δ background were cultured at a nonpermissive temperature (16°C) for 15 h and subjected to microscopic observation (C) or western blotting (D). (C) Left panels: fluorescence images of GFP–Bqt4 (upper panels) and bright-field images (lower panels). Scale bar: 10 µm. Right panel: the fluorescence intensities in the nuclei were quantified and the relative values were plotted. Bars represent the mean±s.d. The numbers of cells analyzed are shown at the bottom. ****P<0.0001 via two-tailed unpaired Student's *t*-test. (D) The protein amounts of GFP–Bqt4 and β-actin were detected by anti-GFP and anti-β-actin antibodies, respectively. Molecular mass markers are shown on the left. β-actin was used as a loading control. Images in B and D are from a single experiment. Images in C are representative of two independent experiments.

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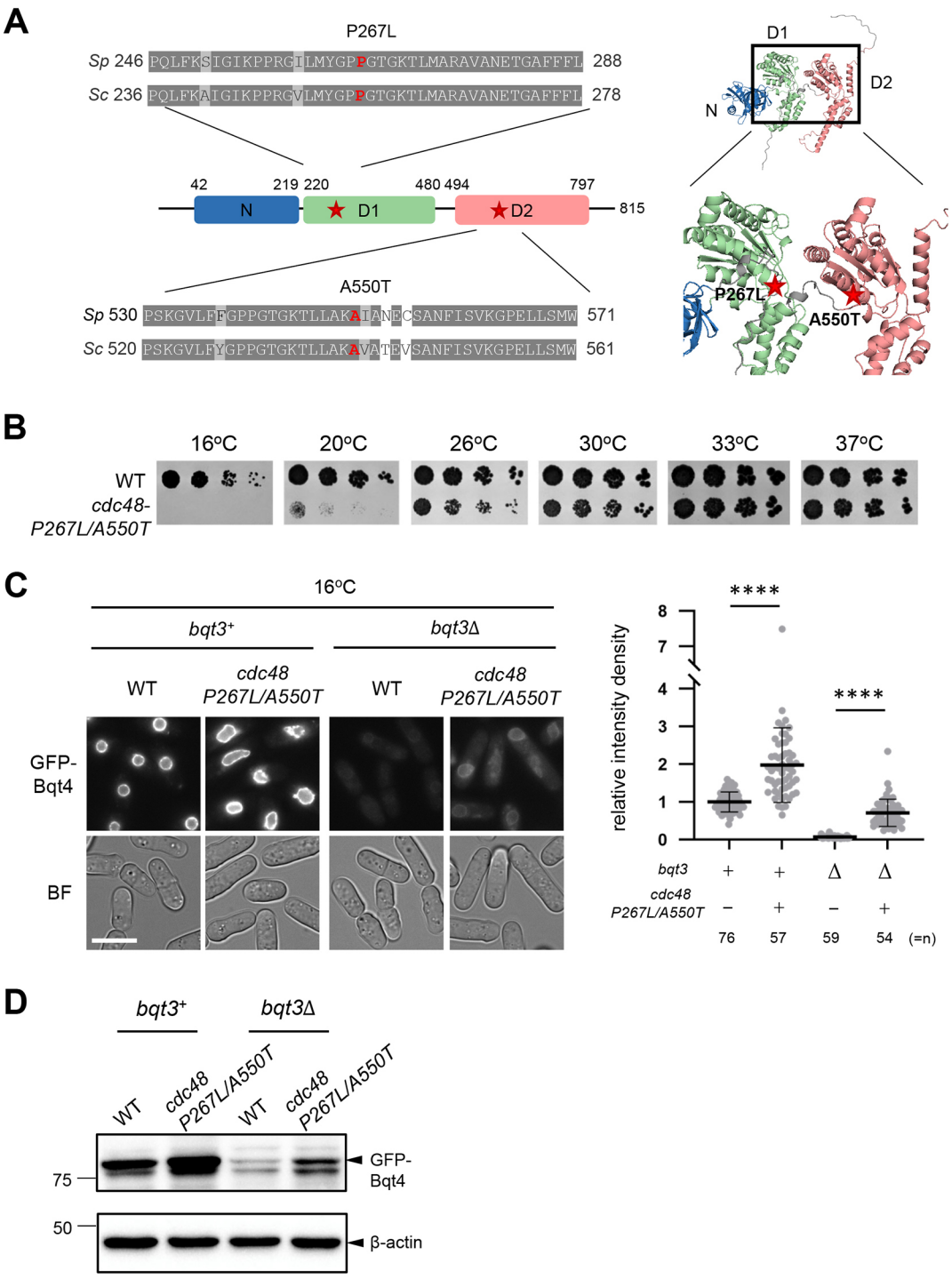

Fig. 5 (original figure). Removal of Bqt4 requires the Cdc48 ATPase complex. (A) The Cdc48 mutant used in this study. Left panel: amino acid sequence alignment around the D1 and D2 domains of *S. pombe* (*Sp*) and *S. cerevisiae* (*Sc*) Cdc48. Gray and light gray shades denote identical and similar amino acids, respectively (top and bottom). The amino acids indicated in red (P267 and A550; corresponding to red stars in the schematic diagram in the middle) are both mutated in this study (P267L/A550T). Right panel: the mutation sites are shown in the predicted three-dimensional structure of Cdc48 by AlphaFold2 (https://alphafold.ebi.ac.uk/entry/Q9P3A7). (B) Temperature sensitivity of *cdc48-P267L/A550T* mutant. Fivefold serially diluted cells harboring *cdc48-P267L/A550T* mutant were spotted on YES plates and cultured at different temperatures as indicated at the top. (C,D) Effect of hypomorphic mutation of *cdc48* on Bqt4 degradation. Cells harboring the *cdc48-P267L/A550T* mutant in the *bqt3*$^{+}$ or *bqt3*Δ background were cultured at a nonpermissive temperature (16°C) for 15 h and subjected to microscopic observation (C) or western blotting (D). (C) Left panels: fluorescence images of GFP–Bqt4 (upper panels) and bright-field images (lower panels). Scale bar: 10 µm. Right panel: the fluorescence intensities in the nuclei were quantified and the relative values were plotted. Bars represent the mean±s.d. The numbers of cells analyzed are shown at the bottom. ****P<0.0001 via two-tailed unpaired Student's *t*-test. (D) The protein amounts of GFP–Bqt4 and β-actin were detected by anti-GFP and anti-β-actin antibodies, respectively. Molecular mass markers are shown on the left. β-actin was used as a loading control. Images in B and D are from a single experiment. Images in C are representative of two independent experiments.

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Fig. S5 has been updated to refer to the Cdc48 mutant as *cdc48-P251L/A544T* throughout the figure and legend. The corrected and original figures are shown below.

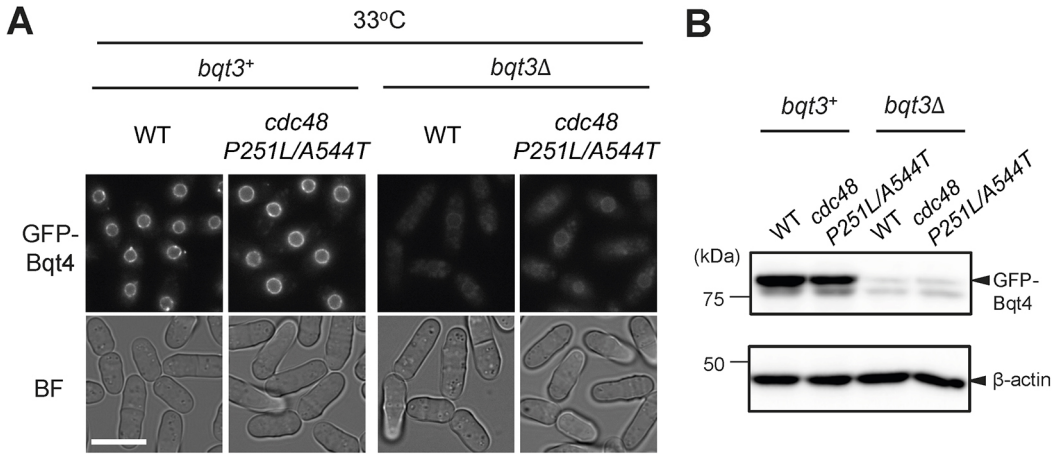

Fig. S5 (corrected figure). Degradation of Bqt4 is not significantly affected in Cdc48 mutant at the permissive temperature. Cells harboring the cdc48-P251L/A544T mutant in the *bqt3+* or *bqt3Δ* background were cultured at 33°C for 15 h and subjected to microscopic observation (A) or western blotting (B). (A) Fluorescence images of GFP-Bqt4 (upper panels) and bright-field images (lower panels). Bar: 10 µm. (B) The protein levels of GFP-Bqt4 and β-actin were detected by anti-GFP and anti-β-actin antibodies, respectively. The molecular weight markers are shown on the left.

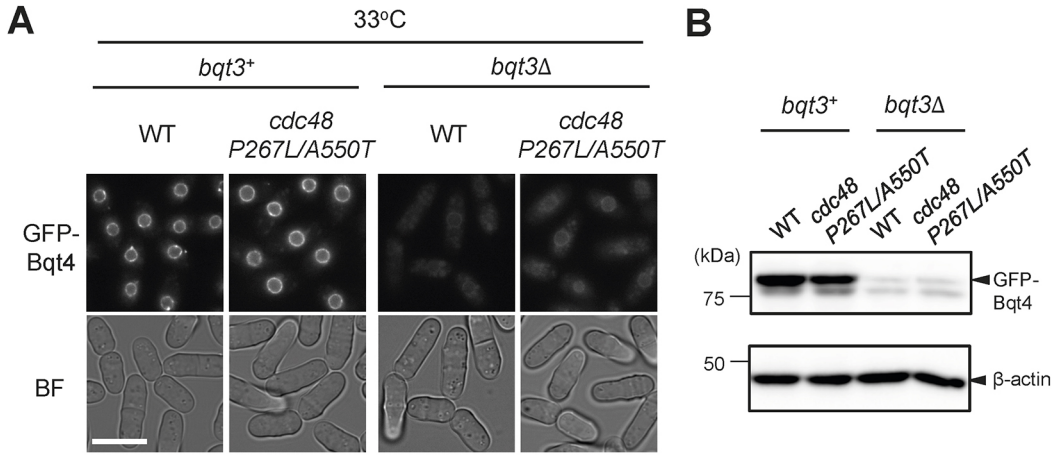

Fig. S5 (original figure). Degradation of Bqt4 is not significantly affected in Cdc48 mutant at the permissive temperature. Cells harboring the cdc48-P267L/A550T mutant in the *bqt3+* or *bqt3Δ* background were cultured at 33°C for 15 h and subjected to microscopic observation (A) or western blotting (B). (A) Fluorescence images of GFP-Bqt4 (upper panels) and bright-field images (lower panels). Bar: 10 µm. (B) The protein levels of GFP-Bqt4 and β-actin were detected by anti-GFP and anti-β-actin antibodies, respectively. The molecular weight markers are shown on the left.

The reporting of experiments using the Cdc48 mutant has been corrected in the 'Degradation of Bqt4 requires the Cdc48 ATPase complex' section of the Results. The original text was as follows:

To this end, we generated a *cdc48* mutant bearing mutations in both the D1 (P267L) and D2 (A550T) ATPase domains based on the temperature-sensitive *cdc48-6* allele in *S. cerevisiae* (Schuberth and Buchberger, 2005; Ruggiano et al., 2016) (Fig. 5A). The *cdc48* mutant (*cdc48-P267L/A550T*) exhibited cold-sensitive growth defects (Fig. 5B), suggesting that these mutations impaired Cdc48 activity in *S. pombe*. Inactivation of *cdc48-P267L/A550T* by shifting to a nonpermissive temperature of 16°C elevated the level of GFP–Bqt4 in *bqt3*$^{+}$ and *bqt3*Δ cells, as observed by fluorescence microscopy (Fig. 5C) and western blotting (Fig. 5D); however, it was degraded at 33°C (Fig. S5), indicating that Cdc48 was required for Bqt4 degradation.

The corrected text now reads:

To this end, we generated a *cdc48* mutant bearing mutations in both the D1 (P251L) and D2 (A544T) ATPase domains based on the temperature-sensitive *cdc48-6* allele (P257L/A540T) in *S. cerevisiae* (Schuberth and Buchberger, 2005; Ruggiano et al., 2016) (Fig. 5A). The *cdc48* mutant (*cdc48-P251L/A544T*) exhibited cold-sensitive growth defects (Fig. 5B), suggesting that these mutations impaired

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Cdc48 activity in *S. pombe*. Inactivation of *cdc48-P251L/A544T* by shifting to a nonpermissive temperature of 16°C elevated the level of GFP–Bqt4 in *bqt3*$^{+}$ and *bqt3*Δ cells, as observed by fluorescence microscopy (Fig. 5C) and western blotting (Fig. 5D); however, it was degraded at 33°C (Fig. S5), indicating that Cdc48 was required for Bqt4 degradation.

Discussion of these results in the 'An INM protein degradation pathway in *S. pombe*' section has also been corrected. The original text was as follows:

In *S. pombe*, Cdc48 mutations at its ATPase active center impair Bqt4 degradation (Fig. 5), indicating that ERAD and/or INMAD degradation involves the Cdc48 complex; however, it remains unclear whether Cdc48 extracts Bqt4 from the INM.

The corrected text now reads:

In *S. pombe*, Cdc48 mutations impair Bqt4 degradation (Fig. 5), indicating that ERAD and/or INMAD degradation involves the Cdc48 complex; however, it remains unclear whether Cdc48 extracts Bqt4 from the INM.

Additionally, Table S1 has been updated to correctly report the genotype of the Cdc48 mutant strains used in the study. The genotype for strain TL311 has been updated from *h*$^{-}$ *lys1-131 cdc48Δ::kan*^{r} *aur1*^{r}*::cdc48p-cdc48-P267L/A550T* to *h*$^{-}$ *lys1-131 cdc48Δ::kan*^{r} *aur1*^{r}*::cdc48p-cdc48-P251L/A544T*; the genotype for strain T306 has been updated from *h*$^{-}$ *bqt4Δ::hph lys1*$^{+}$*::bqt4p-GFP-bqt4 cdc48Δ::kan*^{r} *aur1*^{r}*::cdc48p-cdc48-P267L/A550T* to *h*$^{-}$ *bqt4Δ::hph lys1*$^{+}$*::bqt4p-GFP-bqt4 cdc48Δ::kan*^{r} *aur1*^{r}*::cdc48p-cdc48-P251L/A544T*; and the genotype for strain TL307 has been updated from *h*$^{-}$ *bqt4Δ::hph lys1*$^{+}$*::bqt4p-GFP-bqt4 bqt3Δ::NAT cdc48Δ::kan*^{r} *aur1*^{r}*::cdc48p-cdc48-P267L/A550T* to *h*$^{-}$ *bqt4Δ::hph lys1*$^{+}$*::bqt4p-GFP-bqt4 bqt3Δ::NAT cdc48Δ::kan*^{r} *aur1*^{r}*::cdc48p-cdc48-P251L/A544T*.

The authors apologise to readers for these errors, which do not affect the conclusions of the article.