

The Sec1p/Munc18 (SM) protein, Vps45p, cycles on and off membranes during vesicle transport

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The authors noticed a duplication of data in Fig. 3 B between the panels representing subcellular fractionations of wild-type and *vps21Δ* cells. Given the time that has elapsed since the original date of publication, the original data could not be found to correct the figure, so a new version of Fig. 3 B from a recent repeat of the experiment has been provided below.

The html and pdf versions of this article have been corrected. The error remains only in the print version.

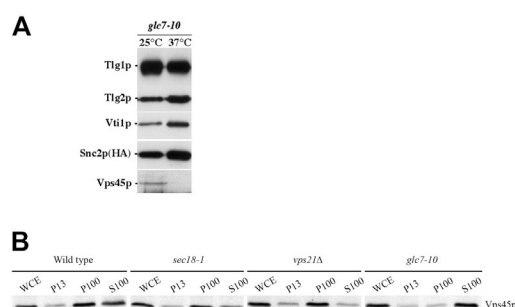


Figure 3. Vps45p loses membrane association upon inactivation of PP1. (A) Tlg1p-containing complexes were immunoprecipitated from cells harboring the *glc7-10* mutation (NOzY23) grown at 25°C (25°C), or grown at 25°C and then incubated at 37°C for 10 min before cell lysate preparation (37°C). Immunoblot analysis was used to detect the amount of Tlg1p, Tlg2p, Vti1p, HA-tagged Snc2p, and Vps45p in the immunoprecipitated complexes. (B) wild-type (SF838–9D), *sec18-1* (NOzY22), *vps21Δ* (SGY79), and *glc7-10* (PAY704–1) cells were incubated at 37°C for 10 min before fractionation by differential centrifugation to yield a whole cell extract (WCE), a low-speed membrane pellet (P13), a high-speed membrane pellet (P100), and a soluble, cytosolic fraction (S100). The amount of Vps45p in each fraction was assessed using immunoblot analysis.