## *Correction*

In the article "Yeast Gga Coat Proteins Function with Clathrin in Golgi to Endosome Transport," by C. Costaguta, C.J. Stefan, E.S. Bensen, S.D. Emr, and G.S. Payne (Mol. Biol. Cell [2001], *12*, 1885–1896), we presented, in Figure 5, the results of pulse-chase immunoprecipitations of Vps10p and Kex2p using a *gga1*Δ *gga2*Δ *vps28*Δ strain (YCS201). The data showed that turnover of Vps10p was delayed in this strain compared with *vps28*Δ cells, leading us to conclude that deletion of the *GGA* genes in a class E *vps* mutant impairs Vps10p transport to endosomes. We have now repeated this experiment with two independently derived *gga1*Δ *gga2*Δ *vps28*Δ strains and observed no delay in Vps10p turnover compared with *vps28*Δ cells. Furthermore, a low-copy plasmid with *GGA2* did not restore rapid Vps10p turnover in the original *gga1*Δ *gga2*Δ *vps28*Δ strain, indicating that the absence of Gga proteins does not account for delayed Vps10p turnover in this strain. Based on these results, we now believe that YCS201 contained an additional defect that impaired Vps10p turnover. These results do not alter our conclusion that Gga proteins play important roles in cargo-selective clathrin-mediated traffic from the Golgi to endosomes because this conclusion is supported by other data in the paper and by results from other publications (Black and Pelham, J. Cell Biol. *151*, 587–600, 2000, Katzmann *et al.*, Cell *106*, 145–155, 2001). Nevertheless, we apologize for any inconvenience caused by this revision. Degradation of Vps10p in class E mutants lacking the Gga proteins could be due to transport of Vps10p from the *trans*-Golgi network to endosomes by an alternative, indirect pathway, or to loss of compartmental distinctions between the *trans*-Golgi network and endosomes. Further experiments are needed to distinguish between these possibilities.