## Correction

The authors of "The alternate AP-1 adaptor subunit Apm2 interacts with the Mil1 regulatory protein and confers differential cargo sorting" (Mol. Biol. Cell [2016] 27, 588–598; originally published in MBoC In Press as 10.1091/mbc.E15-09-0621) wish to make a correction to Figure 4G of the article. In the original HTML and PDF versions, the bottom (horizontal) label incorrectly read " $apm2\Delta$ ." This label has been changed to " $mil1\Delta$ " in the corrected figure below.

The HTML and PDF versions were corrected on the *Molecular Biology of the Cell* website on July 5, 2016. These corrections may not appear on copies of the article that reside on other websites.

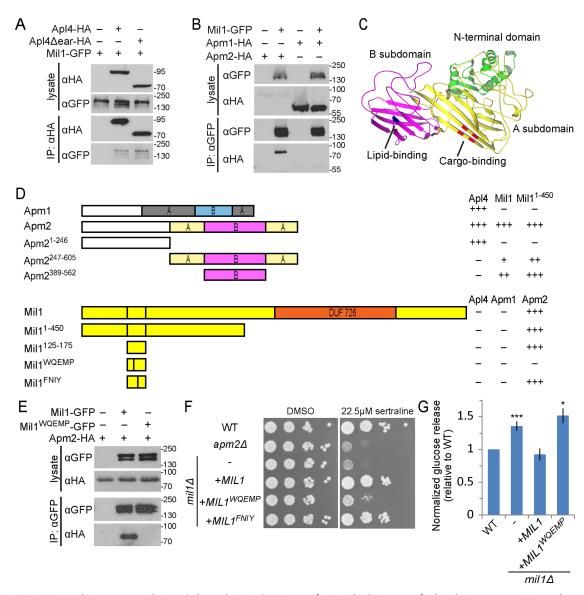


FIGURE 4: Mil1 interacts with Apm2 through its WQEMP motif. (A) Mil1-GFP copurified with immunoprecipitated Apl4-3HA and with a truncated version lacking the  $\gamma$  appendage (Apl4 $\Delta$ ear-HA) known to bind several AP-regulatory proteins. Loading of lysate relative to immunoprecipitate was 1:9. All proteins were genomically tagged. (B) Pull down of Mil1-GFP in strains coexpressing either Apm1-3HA or Apm2-3HA shows that Mil1 binds specifically to Apm2. All proteins were genomically tagged. (C) Phyre2 homology model of Apm2, colored to indicate the N-terminal AP-binding domain (green) and a C-terminal region composed of A (yellow) and B (magenta) subdomains. Key residues predicted to be involved in Yxx $\Phi$  binding (red) or lipid binding (blue) based on alignment with regions of AP-1 and AP-2  $\mu$  subunits are indicated. (D) Yeast two-hybrid mapping of Apm2-Mil1-binding domains. Full-length or truncated Apm2 constructs were fused to the GAL4 DNA-binding domain (GBD), and full-length, truncated, or mutated Mil1 constructs were fused to the GAL4-activating domain (GAD). Qualitative interaction strengths are

indicated. Mil1<sup>WQEMP</sup> represents the W<sup>143</sup>QEMP>AAEAA mutant, and Mil1<sup>FNIY</sup> represents the F<sup>152</sup>NIY>ANAA mutant. (E) Anti-GFP immunoprecipitation of plasmid-expressed wild-type Mil1-GFP or Mil1<sup>WQEMP</sup>-GFP from strains coexpressing genomically tagged Apm2-3HA. (F) Sertraline sensitivity was assessed by plating strains in a 10× dilution series on YPD containing 22.5  $\mu$ M sertraline or DMSO as a control. (G) The  $mil1^{WQEMP}$  mutant is unable to restore sorting of the Snc1 reporter GSS. Cell-surface GSS levels in the indicated strains were determined by quantifying invertase activity and normalizing to levels in wild-type cells. Unpaired t test compared with wild type, \*\*\*p < 0.0001 and \*p < 0.05. Error bars represent SEM (n = 10).