concentrations) overlap. In each case, data from three protein concentrations and one speed are displayed (open symbols) along with the best single ideal species fit (solid lines). The best fits for each protein were derived from global fits to data collected at three concentrations and two speeds. The residuals (differences between the raw absorbance data and the fit are shown below each panel.

Figure 5. Mutational analyses of crystallographic Vps4 dimer interfaces.

(a) Crystallographic Vps4 dimer interfaces. Interface 1 is a symmetric interface between two large ATPase domains (residue Q216 is shown in cyan), Interface 2 is a symmetric interface between two small ATPase domains (residue L407 is shown in blue), and Interface 6 is an asymmetric interface between the large and small ATPase domains (residues L151 and W388 are shown in orange and green, respectively).

(b) Gel filtration chromatograms of $Vps4_{\Delta MIT}$ proteins with the following mutations: Q216A (Interface 1), L407D (Interface 2), L151D (Interface 6), and W388A (Interface 6). $Vps4_{\Delta MIT}$ proteins used here and elsewhere contained the E233Q mutation, which allowed ATP binding but inhibited hydrolysis. For reference, the elution profile of the "wild type" $Vps4_{\Delta MIT,E233Q}$ protein is shown in red in each panel, elution positions for monomeric (1) and dimeric (2) proteins are shown as dotted vertical lines, and the elution positions of molecular weight standards are shown below the chromatograms. Vps4 protein concentrations were 150 μM in all cases. Note that at low micromolar concentrations, the dimeric proteins exhibited concentration-dependent mobilities (not