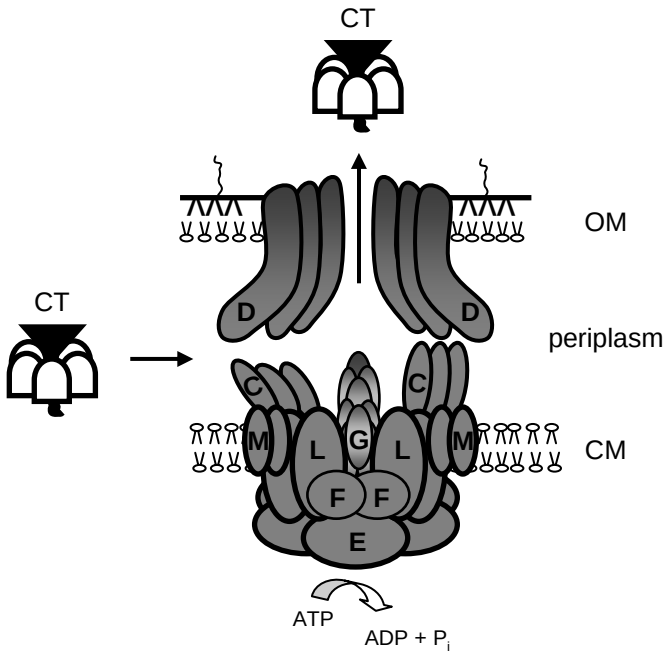

Figure S1. Model of the T2S complex in *V. cholerae*. Cholera toxin (CT) engages the secretion apparatus in the periplasmic compartment. This may lead to conformational changes in the T2S complex that result in polymerization of EpsG into a piston-like structure that extends from the EpsE-F-L-M platform in the cytoplasmic membrane (CM). Through the active process of EpsG polymerization, CT may be pushed through the channel in the outer membrane (OM) in an energy-depended process possibly regulated by EpsE. The number of EpsE and EpsD subunits in the T2S complex is likely six and twelve, respectively. The copynumber of the other components is not known. PilD and the Eps proteins H, I, J, K and N are not shown in this model.

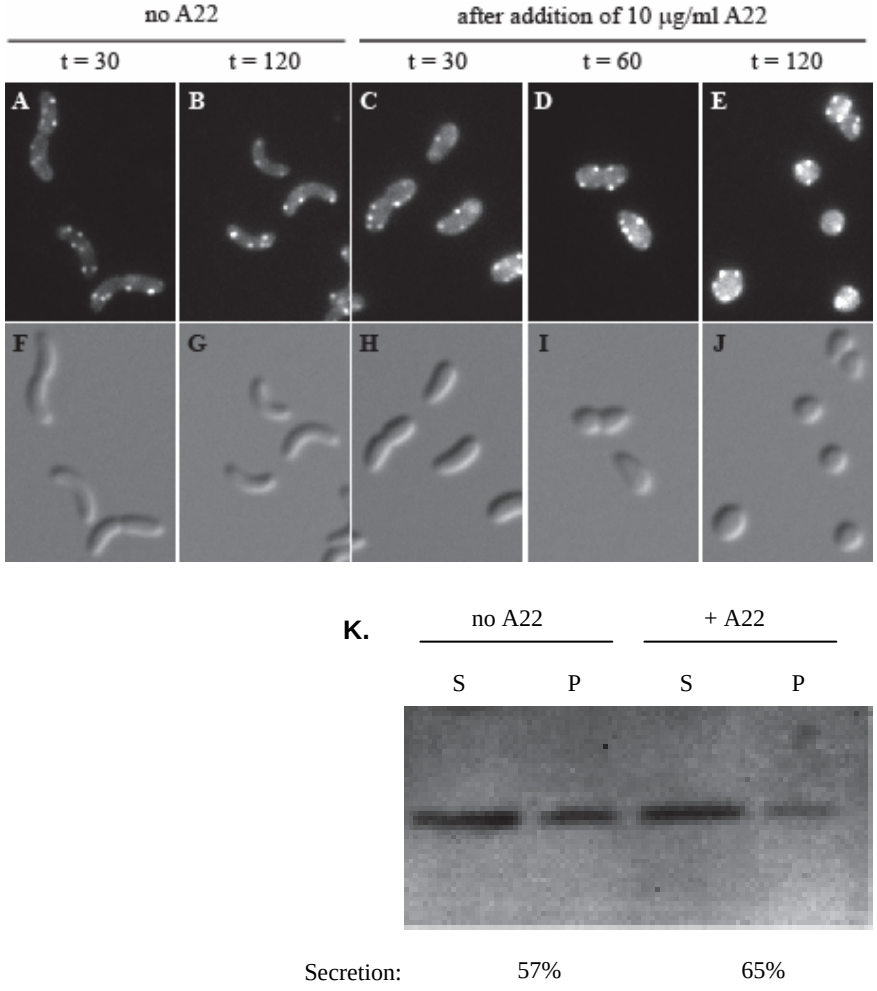

Figure S2. A22 treatment of *gfp-epsC* strain. Log phase cultures of *gfp-epsC* strain containing pMMB68 grown in the absence (A, B, F and G) or presence of 10 mg/ml A22 for 30 (C, H), 60 (D, I), and 120 minutes (E, J) were examined by fluorescence (panels A-E) and differential interference contrast microscopy (F-J). (K) Following growth in the absence (lanes 1 and 2) or presence (lanes 3 and 4) of A22 for 120 min, cells were separated from culture supernatants by centrifugation, and the proteins in both the cell pellets and supernatant fractions separated by SDS-PAGE. The amount of EtxB present in the culture supernatant (S;lanes 1 and 3) and cell pellets (P; lanes 2 and 4) was assessed by immunoblotting and quantified using the Typhoon Trio system and ImageQuant TL software. The numbers below the blot indicate the level of secretion as a percent of total amount of EtxB.