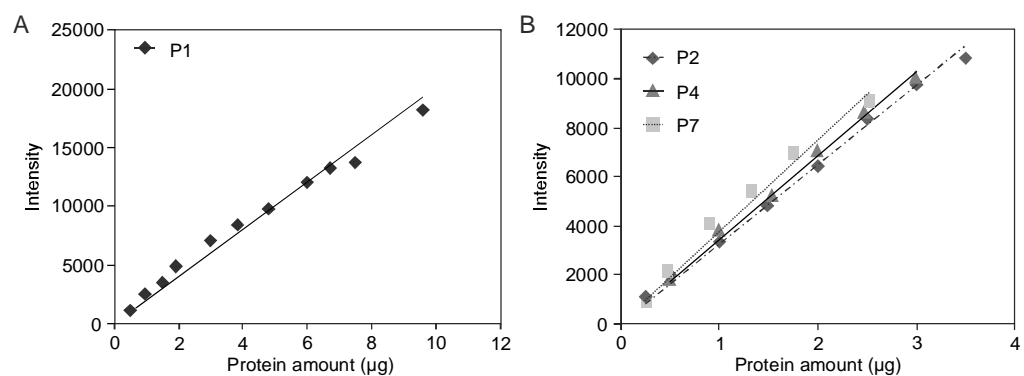
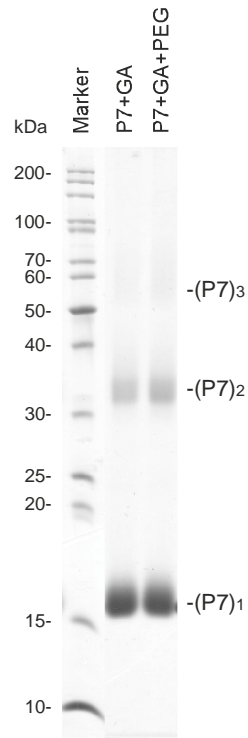


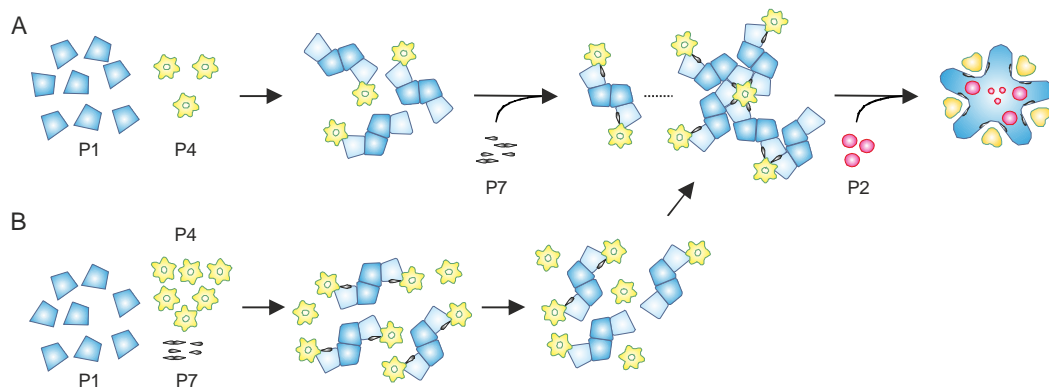
## SUPPLEMENTARY FIGURES



Supplementary Figure 1. Linear relationship between protein quantity and the protein band intensity in a Coomassie-stained polyacrylamide gel for bacteriophage  $\phi 6$  proteins P1 (A), P2, P4 and P7 (B). The parameters of linear regressions were used for stoichiometric analyses.



Supplementary Figure 2. Cross-linking of P7 of bacteriophage  $\phi 6$  with 0.5% glutaraldehyde (GA) in 20mM potassium phosphate pH7.2 buffer in the presence or absence of 6% polyethylene glycol 4000 (PEG). After 30min incubation at room temperature, the cross-linking products were separated by electrophoresis in 15% SDS-polyacrylamide gel electrophoresis. The migration of P7 monomers  $(P7)_1$ , dimers  $(P7)_2$  and trimers  $(P7)_3$  are indicated on the right. The marker is Unstained Protein Ladder (Fermentas).



Supplementary Figure 3. Schematic representation of the *in vitro* assembly pathway of bacteriophage  $\phi 6$  polymerase complex (adopted from Poranen et al. 2001). The two conformers of P1 are shown in light and dark blue. The ring-like (yellow) and drop-like (grey) shapes correspond to the P4 hexamer and P7, respectively. (A) During procapsid assembly, a  $(P1)_4(P4)_6$  nucleation complex is formed in the absence of P7. In the presence of P7, the rate of assembly is increased and the formation of a  $(P1)_4(P4)_{12}(P7)_n$  complex becomes rate limiting. Recent data by Nemecek et al. (2012) propose that P7 is located at the interface of the P1 conformers surrounding the P4 hexamers (light blue), thus P7 could stabilize the formation of the precursor for the five-fold symmetry vertex of the PC. P2 is incorporated interior of the growing P1 shell before the assembly of the shell is completed. (B) In the presence of excess of P4, the rate of assembly is decreased due to the accumulation of  $(P1)_4(P4)_{12}(P7)_n$  complexes which cannot interact with each other to form the shell.

## References

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