with the 5′ domain. These interdomain interactions are absent in state A but start to emerge in the subsequent states.

In states A and B, the ribosome assembly factor Krr1 binds to the central domain at the same position as h45 does in the mature SSU, preventing its assembly. Concomitant with removal of helix H6 of the 5′ ETS, Krr1 and its binding protein Faf1, which bind to H6, are released in state C, allowing for integration of h45 (Fig. 3, A to C, and fig. S7A). In addition, Utp7, Mpp10, Utp12, Utp21, and Imp4, which are located near H6, become partially unstructured in state C, and Imp3, in complex with an a-helix of Mpp10, rotates by ~15° (Fig. 3B). Moreover, h45 and its binding protein Pno1, anchored on the UTPB complex, rotate as a whole unit by ~60°, which brings h45 closer to its binding site in the central domain. The movement of h45 also depends on prior removal of H6 because the new position of h45 was previously occupied by H6 and the H5 to H6 linker (Fig. 3A). Removal of H6 thus appears to drive maturation of the central domain.

Helix E of ES6 (ES6E) in the central domain of 18S rRNA makes a long-range interaction with helix B of ES3 (ES3B) in the 5′ domain in mature SSU (Fig. 1A and fig. S5A). ES6E initially binds Utp22 in state A and is resituated to bind Utp20 in state B (Fig. 3C and fig. S6, A to C). The structural remodeling brings ES6E closer to, but not yet in contact with, the 5′ domain (Fig. 3E). The original position of ES6E on Utp22 is occupied by the TPR domain of Rrp5, suggesting that Rrp5 drives the remodeling of ES6E. Rrp5 additionally contains 12 S1 RNA-binding domains, some of which can be visualized (fig. S6, B to D). The TPR domain of Rrp5 was mistakenly modeled in previous state 1 because of insufficient classification.

In state D, Kre33, Enp2, Lcp5, and a short segment of Brf2, which are bound to the apical part of the 5′ domain in earlier states, are missing (Figs. 2, D and E, and 3, C and D). Release of these AFs is probably facilitated by prior release of Krr1 in state C because the extended C-terminal tail of Krr1 binds Enp2 and Bfr2 (Fig. 3C). Utp20 is om97.2e n