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IN BRIEF

SIGNAL TRANSDUCTION

Cooperative assembly of TGF-β superfamily signaling complexes is mediated by two disparate mechanisms and distinct modes of receptor binding.

Groppe, J. *et al. Mol. Cell* **29**, 157–168 (2008)

Signalling by the transforming growth factor-β3 (TGFβ3) requires binding of the high-affinity TGFβ receptor II (TβRII); the ligand-bound TβRII subsequently recruits the low-affinity TβRI. Groppe *et al.* now report the X-ray structure of a TGFβ3 ligand dimer in a hexameric complex with the extracellular domains of TβRI and TβRII pairs. An N-terminal extension in TβRII tethers TβRI to the ligand, and a five-residue finger in TβRI that forms hydrogen bonds with an Asp residue in TβRII is crucial for cooperative hexameric complex assembly and signal transduction. This mechanism is distinct from the structurally similar bone morphogenetic protein ligand–receptor assembly, which is largely based on avidity.

CELL CYCLE

Centromeric Aurora-B activation requires TD-60, microtubules, and substrate priming phosphorylation.

Rosasco-Nitcher, S. E. *et al. Science* **319**, 469–472 (2008)

Aurora-B kinase is part of the chromosomal passenger complex (CPC), which controls processes such as chromosome congression, kinetochore–microtubule attachments and spindle checkpoint signalling during mitosis. The authors report that Aurora-B activation *in vitro* requires two cofactors: microtubules and telophase disc-60kD, an inner centromere protein that is required for centromeric targeting of the CPC and of haspin kinase activity. Aurora-B can also be regulated by a second mechanism — its substrates can inhibit the kinase activity, which is relieved by substrate phosphorylation by the centromeric kinases PLK1 and haspin. The interplay of substrate inhibition (and relief by mitotic kinases) and cofactor availability may control Aurora-B activity at different times and in different places during the cell cycle.

PROTEIN DEGRADATION

A multimeric assembly factor controls the formation of alternative 20S proteasomes.

Kusmierczyk, A. R. *et al. Nature Struct. Mol. Biol.* 17 Feb 2008 (doi:10.1038/nsmb1389)

Crystal structure of a chaperone complex that contributes to the assembly of yeast 20S proteasomes.

Yashiroda, H. *et al. Nature Struct. Mol. Biol.* 17 Feb 2008 (doi:10.1038/nsmb1386)

The 20S proteasome is a barrel of rings composed of α- and β-subunits and, once assembled, it associates with a 19S regulatory particle (RP) to form the functional proteasome. Two studies now identify two chaperone complexes that are involved in proteasome assembly in budding yeast. Kusmierczyk *et al.* identified Pba3–Pba4, which interacts with α-subunits of the 20S proteasome. Deletion of Pba3–Pba4 causes the formation of altered 20S proteasomes, which also affects RP assembly. Yashiroda *et al.* report the crystal structure of Dmp1–Dmp2 (also known as Poc3–Poc4), which functions early in 20S proteasome assembly and is structurally and functionally similar to the mammalian proteasome-assembly chaperone PAC3.