

Common mechanisms for viral DNA replication

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How DNA replicates is a critical question for understanding life. DNA replication remains difficult to investigate in eukaryotes, where it occurs within the confines of the double-membrane nucleus.

However, the process is far easier to study in the much simpler virus. In simian virus 40 (SV40), the large T-antigen protein (T-ag) is responsible for recognizing DNA sequences required to start replication, called the origin of replication. SV40 T-ag can also cause DNA to melt or unwind.

In a new study published online in the open access journal *PLoS Biology*, Gretchen Meinke, Andrew Bohm, and colleagues report the crystal structure of the DNA-binding domain of SV40 T-ag on a DNA fragment derived from the viral origin of replication.

The structure shows that although T-ag and its functionally analogous protein, papilloma virus E1, share no detectable sequence homology in this region, the two domains bind the DNA in similar ways. In both cases, DNA binding is thought to initiate assembly of a complex of the full-length proteins on DNA. Interestingly, SV40 T-ag DNA-binding domains do not interact with one another when bound to DNA. In addition to describing the molecular details of the DNA–protein interactions and the alterations in protein structure induced by DNA binding, we present a model describing the subsequent assembly events.

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