The novel aspect of the present work on Cut4P is that, a compoment of anaphase promoting complex (APC) or cyclosome, which plays a role in metaphase-to-anaphase transition, is also involved in several other functions like mating type silencing, centromere stability and silencing. Surprisingly, its suppression of ts⁻ phenotype of *esr1*⁻ mutation by *pol*-α gene suggests that paradoxically Cut4 may functions upstream of *DNA pol* α (which is involved in lagging strand synthesis during DNA replication). It is possible that *cut4*⁻ mutation indirectly delays DNA replication due to the delayed mitosis and this defect is overcomed by expression of *DNA polymerse* α. Its genetic interaction with *clr1-clr4* suggests that it may also be physically recruited to perform a direct role in silencing at *mat* loci and centromere loci, in addition to its role in metaphase-to-anaphase transition. Thus, *cut4* performs multiple, pleotropic functions. It would be interesting to check whether *cut4* interacts with *DNA pol-* α, *clr1-clr4* or *swi6* physically *in vivo.*

Cut4P has another interesting characteristic: its effect on establishment of silent epigenetic state at *mat* loci is brought about by acquisition of alternate strucutre, by the protein itself. In its wild type form it causes silencing while the altered form abrogates silencing. Further, the altered form has a prion-like behaviour, that is, it can impart the same structure to other molecules with normal conformation. The altered form, which is inherited cytoplasmically, can either function directly by an altered binding to other silencing factors (*clr1-clr4, swi6*) and/or *mat* loci, or, indirectly, by affecting the properties or pattern of expression of other silencing factors.

This is the first time that a role of prion phenomenon in governing the expression of epigenetic chromosomal target has been shown. Further, studies shall help us unravel the mechanism by which *cut4* exerts its novel effects including the centromere functions, mating type silencing and the basis for its prion-like influence on silencing.