region strongly affects the rate-limiting step of the ATPase cycle of Dictyostelium myosin. Little is known about the nature of this rate-limiting step. In skeletal muscle S1, the hydrolysis of  $ATP^{23}$  or a subsequent conformation change preceding P<sub>i</sub> release<sup>22</sup> is thought to be rate-limiting. It may be that the 50 K/20K junction region modifies the ATPase activity of Dictyostelium myosin by modulating its interaction with actin. For instance, the 50K/20K junction may modulate the ATPase rate by affecting the rate of transition from the weak to the strong binding state. If this is the case, it is reasonable that the sliding velocity, which is related to the strongly bound state, does not change in accordance with the change in ATPase activity. It is not known why all four chimaeric myosins move more slowly than wild-type myosin. We are now modifying our system to express soluble fragments of the chimaeras to measure their kinetic parameters in more depth and allow us to determine more precisely how the 50K/20K junction region modifies the kinetics of the ATPase cycle. 

Received 25 October 1993; accepted 6 January 1994.

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ACKNOWLEDGEMENTS. We thank H. Warrick for reviewing the manuscript. This work was supported by grants from the NIH to J.A.S. T.Q.P.U. was supported by a fellowship from the American Heart Assocation, California affiliate. K.M.R. is a trainee of the Medical Scientist Training Program at the Stanford University School of Medicine.

## CORRECTION

# **Destabilization of tracts of simple** repetitive DNA in yeast by mutations affecting DNA mismatch repair

### Micheline Strand, Tomas A. Prolla, R. Michael Liskay & Thomas D. Petes

Nature 365, 274-276 (1993)

THE penultimate sentence in the legend to Table 1 of this letter should read: "The plasmid pSH91 contains an in-frame insertion of poly(GT) (33 bp) in URA3; this plasmid is identical to pSH44 (ref. 8) except for the orientation of the tract", and not that the plasmid is identical to pSH36 apart from the size of the tract, as originally stated. This error does not affect our conclusions.

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