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Trimethylamine *N*-oxide suppresses the activity of the actomyosin motor

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ABSTRACT

Background: During actomyosin interactions, the transduction of energy from ATP hydrolysis to motility seems to occur with the modulation of hydration. Trimethylamine *N*-oxide (TMAO) perturbs the surface of proteins by altering hydrogen bonding in a manner opposite to that of urea. Hence, we focus on the effects of TMAO on the motility and ATPase activation of actomyosin complexes.
Methods: Actin and heavy meromyosin (HMM) were prepared from rabbit skeletal muscle. Structural changes in HMM were detected using fluorescence and circular dichroism spectroscopy. The sliding velocity of rhodamine-phalloidin-bound actin filaments on HMM was measured using an in vitro motility assay. ATPase activity was measured using a malachite green method.
Results: Although TMAO, unlike urea, stabilized the HMM structure, both the sliding velocity and ATPase activity of acto-HMM were considerably decreased with increasing TMAO concentrations from 0–1.0 M. Whereas urea-induced decreases in the structural stability of HMM were recovered by TMAO, TMAO further decreased the urea-induced decrease in ATPase activation. Urea and TMAO were found to have counteractive effects on motility at concentrations of 0.6 M and 0.2 M, respectively.
Conclusions: The excessive stabilization of the HMM structure by TMAO may suppress its activities; however, the counteractive effects of urea and TMAO on actomyosin motor activity is distinct from their effects on HMM stability.
General significance: The present results provide insight into not only the water-related properties of proteins, but also the physiological significance of TMAO and urea osmolytes in the muscular proteins of water-stressed animals.