Tropomyosin and tonus in lamellibranch adductor muscles

Smooth lamellibranch adductor muscles are able to remain in the contracted state for long periods of time with very little energy expenditure. In addition to a conventional contractile apparatus they may possess a special "catch" mechanism[1], resisting stretch by "setting" the muscle fibre in the contracted state, though in the light of recent work by LOWY *et al.*[2-4], this "set" may be assumed to implement the tetanic activity of the muscle under such conditions. Some evidence presented here suggests that unlike the active contraction of muscle, the "catch" may be associated not with the actomyosin system but with a type of tropomyosin (TMA) first isolated by BAILEY[5,6] from oyster and *Pinna nobilis*.

TMA, so plentiful in adductor muscles and located in definite fibrils[7], known as paramyosin[8], has no ATPase activity and none of the other biological activities characteristics of actin, myosin or of actomyosin. These latter proteins are also present in adductor muscles and sufficiently abundant by themselves to account for the Salyrgan-sensitive active contraction of glycerinated muscle fibres in presence of ATP[9]. TMA may thus have a function different from that of actomyosin, and for its

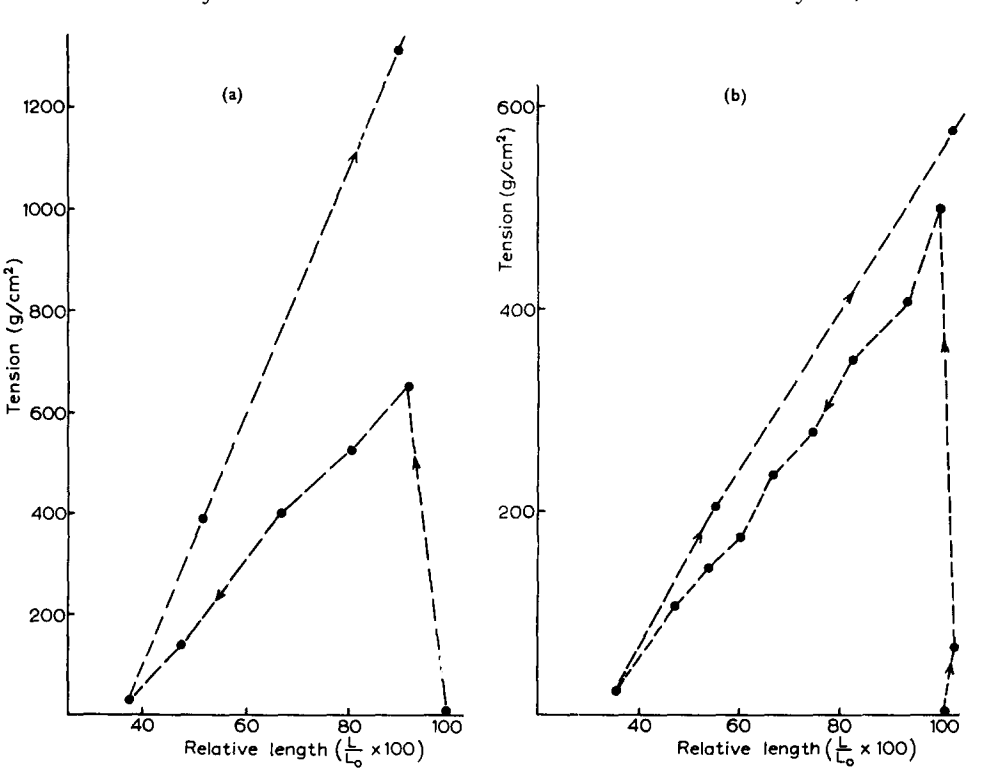

Fig. 1. Tension-length diagrams of glycerinated fibres from the smooth adductor of *Pecten maximus* in presence of 0.005 *M* ATP, 0.004 *M* Mg^{++}, I 0.15, pH 7. (a) Tropomyosin-rich fibre (b) tropomyosin-poor fibre. The fibre is first made to contract isometrically with ATP and is then shortened by a series of quick releases. The points of the lower curve represent the active recovery of tension after quick release at various muscle length and those of the upper curve represent the passive tension after stress-relaxation.

Abbreviations: ATP, adenosine triphosphate; TM, tropomyosin; AM, actomyosin.

elucidation the mechanical properties of TM-rich and TM-poor glycerinated fibres, recently found to co-exist in the smooth adductor of *Pecten maximus*[10], have been compared.

In the TM-rich fibre (AM/TM, 1 : 4) the resistance to stretch for any given muscle length is very much larger than the actively produced tension at the same length, the difference between active and passive tension in the tension-length diagram (*i.e.*, the hysteresis) (Fig. 1a) being attributed to muscle rigidity. In the TM-poor fibre (AM/TM, 1 : 1), investigated under identical conditions, there is much less difference between active tension and resistance to stretch at comparable muscle lengths (Fig. 1b).

The possibility that most of the passive resistance to stretch in the presence of ATP and Mg^{++} is due to an active development of tension by the actomyosin may be ruled out, for the passive tension is still large and barely altered after addition of sufficient Salyrgan to inhibit the myosin ATPase, and thus the tension produced by actomyosin. It might of course be objected that part of the passive resistance to stretch in this case is due to the action of Salyrgan itself on actomyosin, since prolonged treatment is known to denature and to decrease the plasticising effect of ATP[11]. However, in the passively-stretched fibre poisoned with Salyrgan the tension decays at once if Mg-ATP is replaced by free ATP (0.005 *M*). This latter is not a better plasticiser for Salyrgan-treated actomyosin systems than Mg-ATP, but it is in fact better for artificial TM_A threads. Moreover, actomyosin can be selectively denatured in the muscle fibre by pretreatment with ethanol and ether, and yet the passive resistance to stretch of such a fibre can be made to decay by ATP in much the same way as that of an artificial TM_A thread. In such a system as this, ATP can only have influenced the structural protein which remains undenatured, *i.e.*, TM_A.

Other experiments[10] have shown that TM_A binds ATP, and the plasticising effect may thus be interpreted in terms of a decrease in protein–protein interaction due to an increase in net charge. The effect is counteracted by divalent cations such as Ca^{++} and Mg^{++} which combine with ATP. Extrapolating these findings to the behaviour of intact muscle it seems possible that the rigidity of the glycerated fibre in presence of ATP and Mg^{++} corresponds to the tonus or "catch" of tonic muscle, and that free ATP may act as a physiological plasticizer, inducing changes in muscle "viscosity" by affecting the electrostatic machinery of the paramyosin fibrils.

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