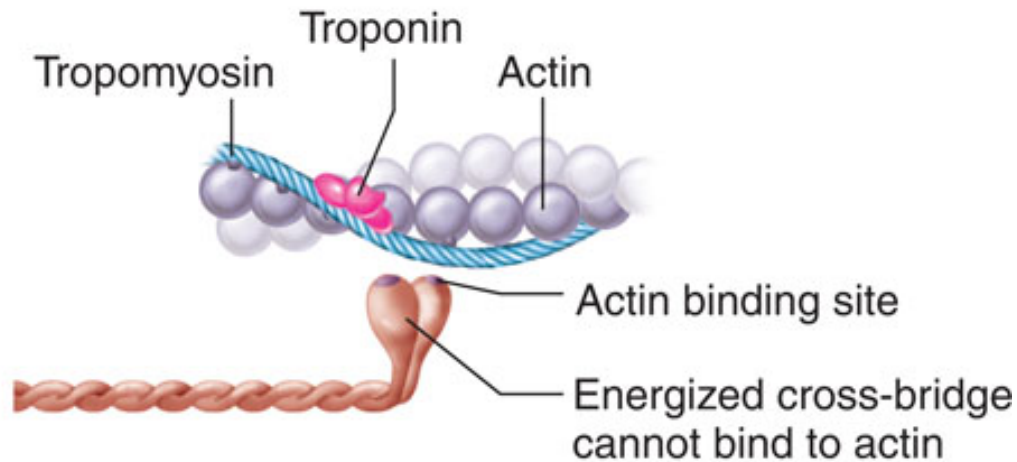


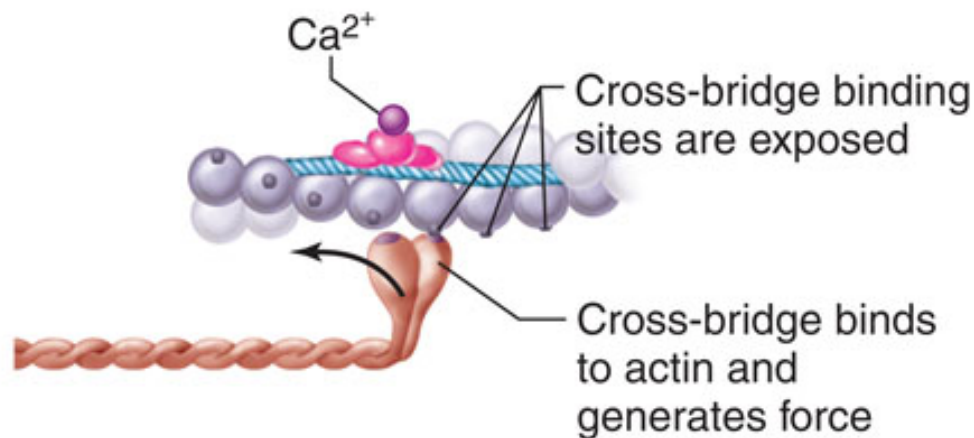
Figure 12-11 The contractile force of skeletal muscle increases in a Ca^{2+} -dependent manner as a result of binding of Ca^{2+} to troponin C and the subsequent movement of tropomyosin away from myosin binding sites on the underlying actin molecules. See text for details. (From MacLennan DH et al: J Biol Chem 272:28815, 1997.)

(a) Low cytosolic calcium, relaxed muscle



VSL[10] 9-9

(b) High cytosolic calcium, Activated muscle



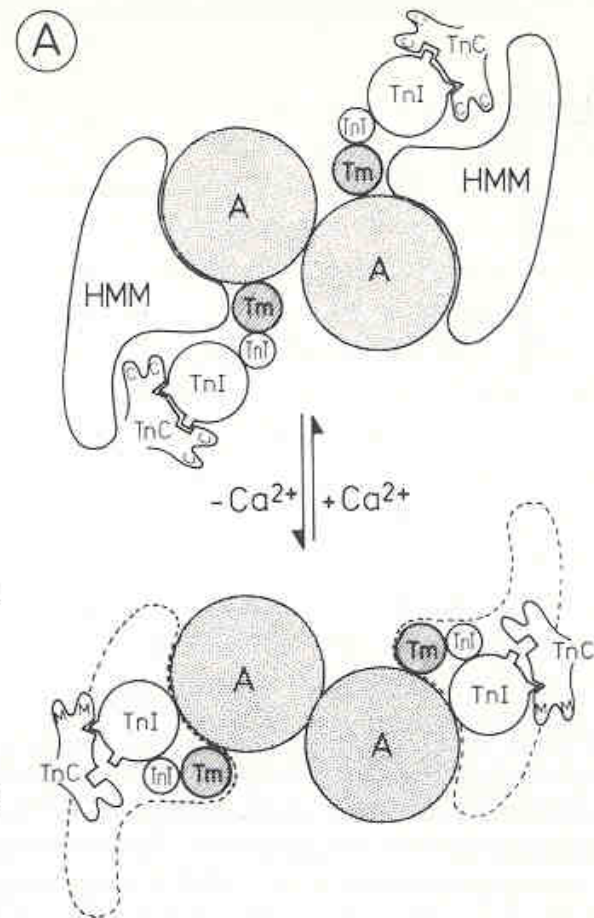
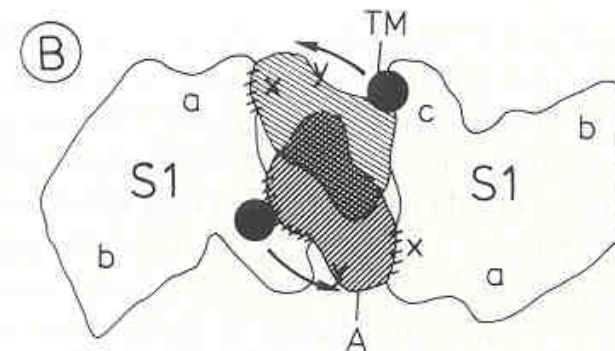


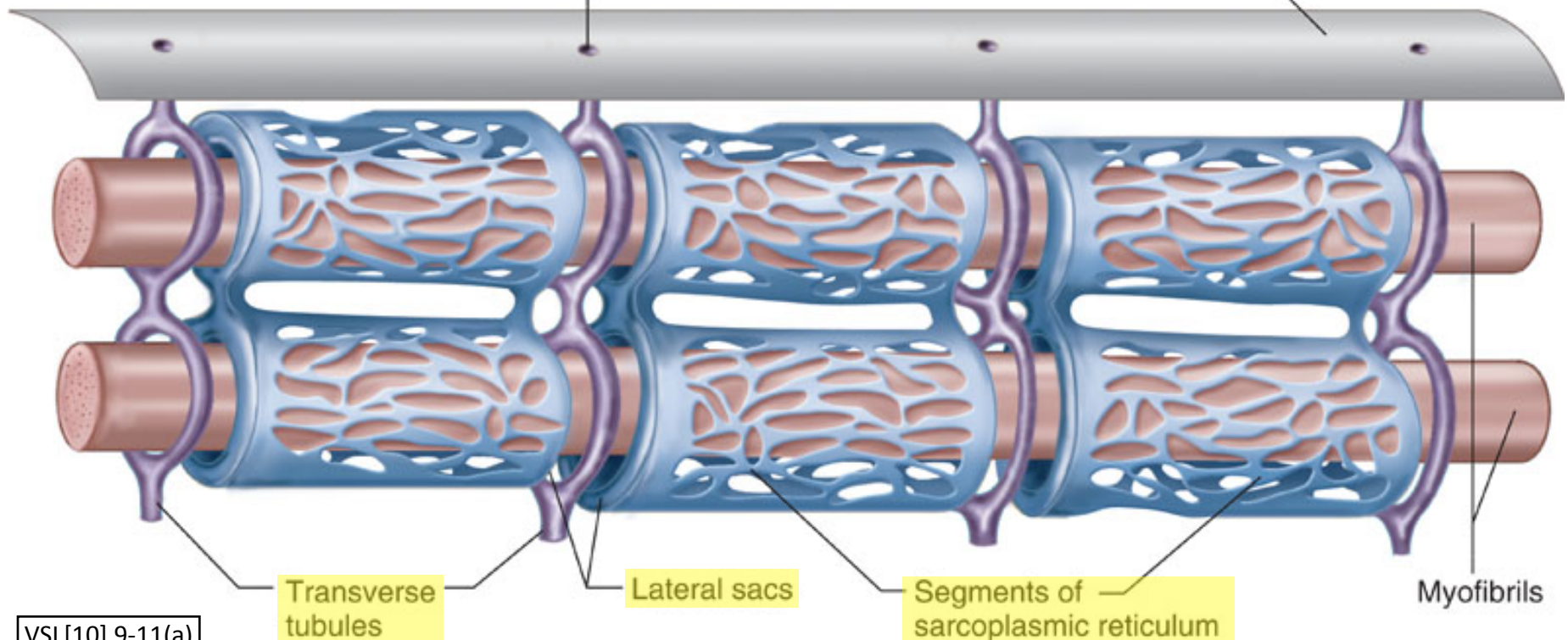
Fig. 4.4 A Model of regulation of skeletal muscle contraction showing the different interactions of troponin subunits, tropomyosin, HMM and actin at high (10^{-5}M) and low (10^{-7}M) concentration of Ca^{2+} . Upon Ca^{2+} activation troponin-C interacts more strongly with troponin-I, while bonds between troponin-I and actin are loosened. At the same time, tropomyosin moves towards the groove between the two actin strands, and crossbridges (HMM) attach to actin. Note that troponin-T interacts with both tropomyosin and troponin-I and possibly also with troponin-C (not shown). (After El-Saleh et al. 1986). *B* A more realistic diagram of the “cross-section” through a thin filament showing the interaction of subfragment-1 (S_1), tropomyosin (TM) and actin (A) in active muscle. Arrows indicate the direction of tropomyosin movement into the “off-position” (x or y) when the muscle relaxes. The subfragment-1 on the left lies 2.7 nm above S_1 on the right. (After Egelman 1985)



(a)

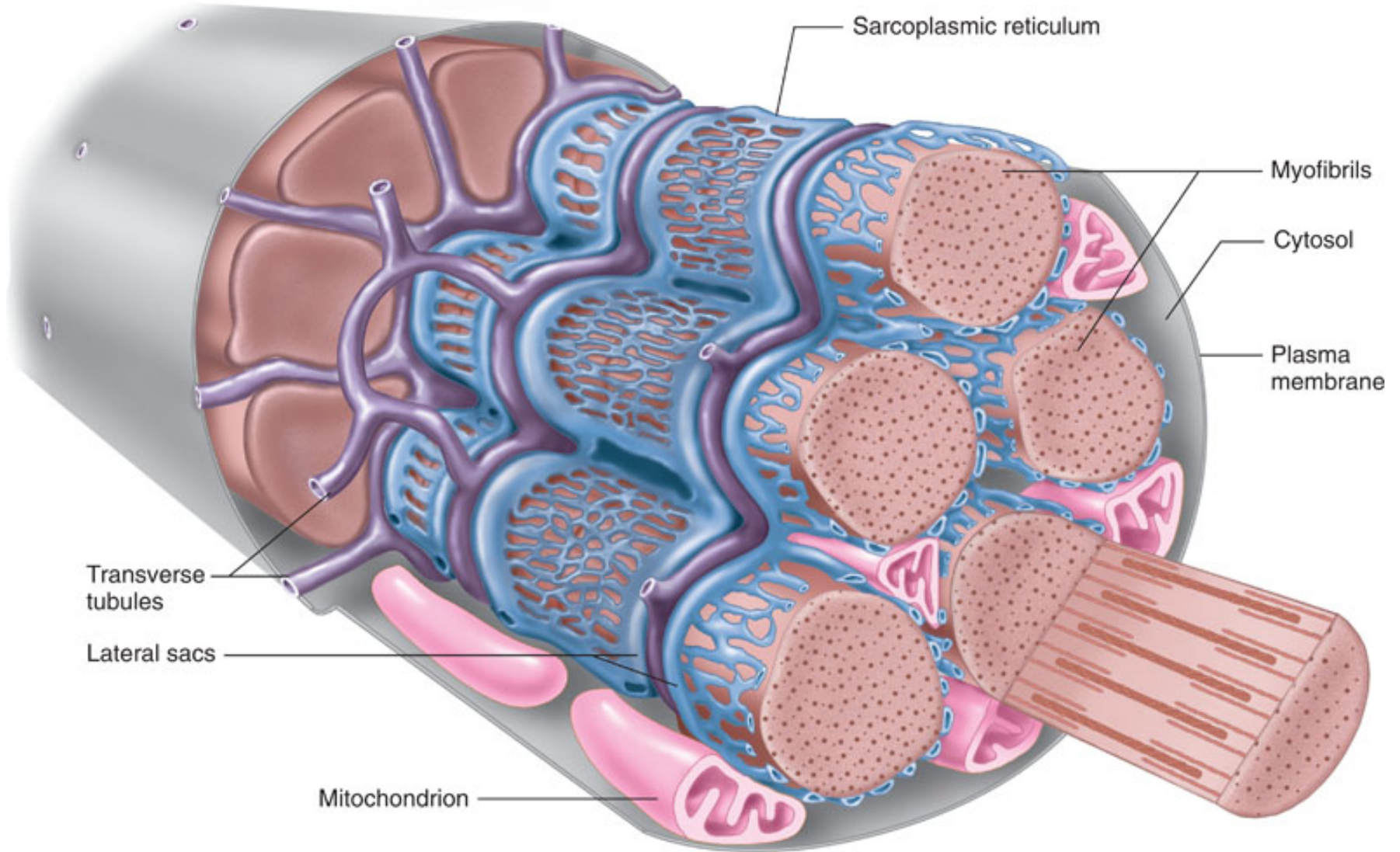
Opening of transverse
tubule to extracellular fluid

Muscle fiber plasma membrane

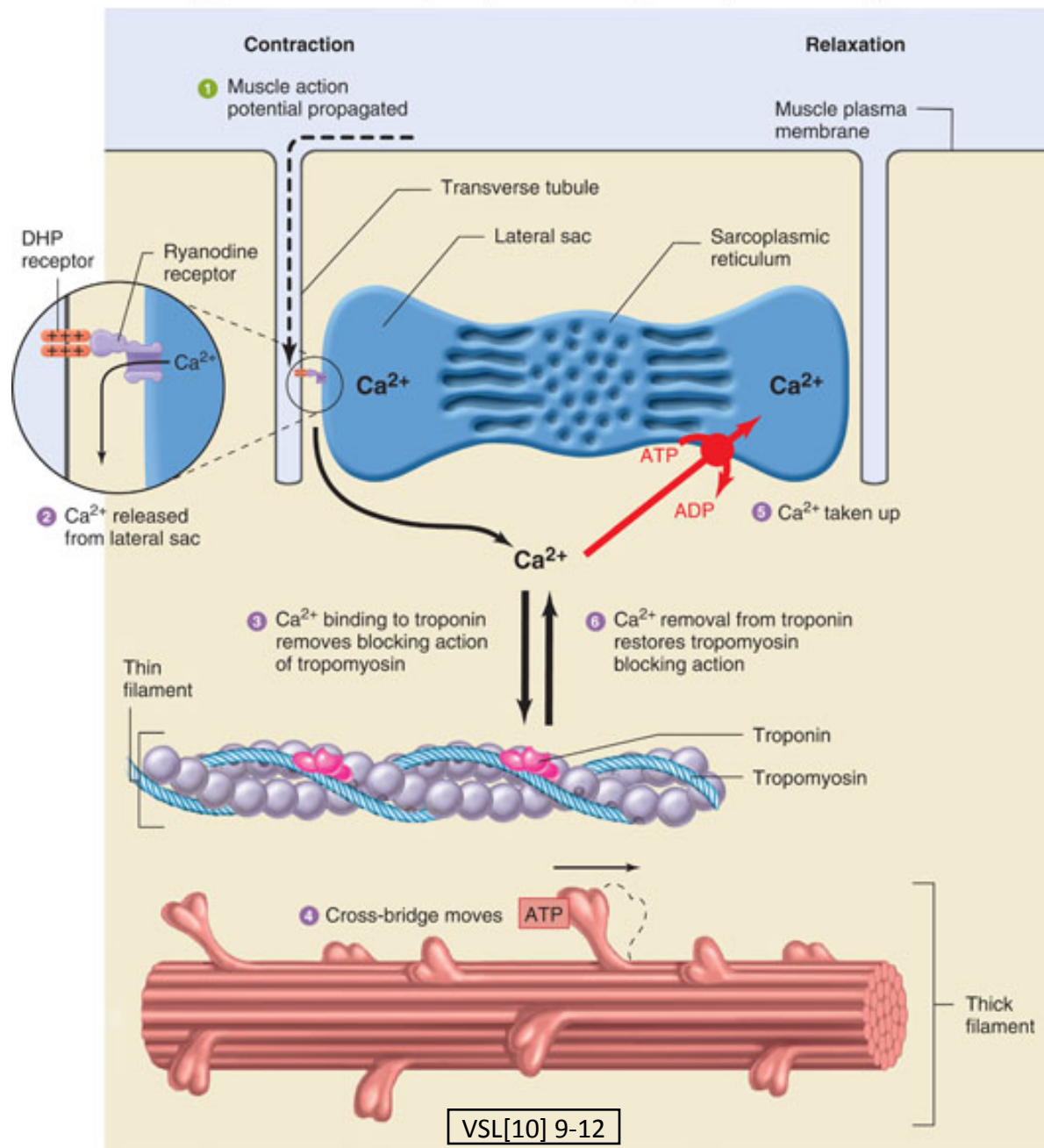


VSL[10] 9-11(a)

(b)



VSL[10] 9-11(b)



END

Video 4, Module 3