tactoid geometry may simply be explained as follows: the distribution of acidic residues of a TM chain has approximate mirror symmetry about a point $\sim70\text{Å}$ from the N terminus. The assembly of the tactoids may not be controlled solely from packing considerations, but may be dominated by the bridging effect of Mg^{2+} between acidic moieties of oppositely directed molecules. If TM molecules were optimally packed in the positively and negatively stained tactoids, there is still no *a priori* reason to believe that $P/4$ should be equated to the observed 28 Å axial period.

Even with an axial pseudo-period of about 19.5 residues in acidic and in apolar residues not in series I and II (refs 5 and 6), azimuthally-identical pseudo-equivalent actin–TM interactions could only occur if an actin separation along one strand ($\sim2\times19.5$ residues) corresponds to a multiple of P' (unless the two chains of TM are homologous and in axial register, in which case actin separations could correspond to a multiple of $P'/2$). P' is the supercoil pitch length measured along the helical axis of the supercoil. On packing grounds, it has been suggested that the two chains of TM may have a relative axial stagger of 14 residues[6-8] thus facilitating head-to-tail assembly of TM molecules, but recent evidence has cast doubts on this conclusion[9,10].

Irrespective of the relative chain stagger in TM, the length and flexibility of side chains may enable the first and/or second chain of TM to make pseudo-equivalent interactions with an actin strand even if precise azimuthal relationships are not maintained (see, for example, Hulmes *et al*.[11] for collagen).

Theoretically, the pitch length of α-fibrous proteins may be found directly using a measurement of the axial separation of the equatorial and near equatorial reflections in the X-ray diffraction patterns[12]. Inherent accuracy, however, is usually low as a result of partial overlapping of the relevant data. Also the possibility that axial sampling moves the maxima of the molecular transform cannot be completely eliminated[13]. P has been estimated for α keratin[14] (140–170 Å); paramyosin[4,15] (136–140 Å and 178 Å) and honeybee silk[16] (140 Å), but not for TM. If the head-to-tail assembly of TM molecules in the grooves of the thin filament form a regular supercoil and troponin has an invariable azimuthal relationship with TM and actin, then $nP = 410 \pm 4$ Å (ref. 17) where n is integral. If $n = 3$, then $P = 137$ Å and there would be six half-turns of supercoil associated with seven actin molecules. As McLachlan and Stewart (private communication) have also pointed out, TM would then present pseudo-equivalent aspects to all seven actins with which it makes contact, as $3P = 7P'/2 = 410$ Å where $P'/2 \sim 2\times19.5$ residue translations

of an α helix or, equivalently, an actin separation.

The pitch length of an α-fibrous coiled coil cannot be continuously reduced without a concomitant increase in the deformation of bond lengths and angles. The point at which the coiled coil structure reaches a minimum energy state is not known, although it is tempting to speculate that this may occur when P is about 140 Å, a value experimentally noted for several α-fibrous proteins.

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LONGLEY REPLIES—In calculating an example of his model of the coiled coil, Crick[1] started with a straight α helix having 18 units in 5 turns, giving a coiled coil with 126 residues in a pitch of 186 Å. If, instead one starts with the very similar helix of 11 units in 3 turns, the resulting coiled coil has 77 residues in a pitch of 114 Å. (The 11/3 helix is one of the alternatives considered by Pauling and Corey[2] and is the structure automatically formed when an α helix is constructed of CPK molecular models.) Thus, large variations in the pitch of a double helix can result from small changes in the α helix from which it is derived, and there is no reason why the pitch of tropomyosin (TM) should be the same as that of paramyosin, keratin or bee silk. The main resistance to bending the α helix into a tighter coiled coil is in stretching and compressing the H bonds[3]. To bend it into a helix of pitch 114 Å would require 0.1–0.2 kcalorie mol^{-1} which is not large, and would strain the H bonds by only 2 or 3%. The observed spread in H-bond lengths is about 8% (ref. 4).

Now, 77 is an odd number so that each

alternate half-turn of such a double α helix would have a different pattern of side groups from the next. This, together with the 14-residue stagger, could be accommodated in the thin filament of muscle if the pattern of contacts between TM and actin alternated (and perhaps commutated) along the length of the TM molecules. In the 'tactoids' likewise, cross linking (by divalent cations) would be simpler if the chemical repeat were compatible with the helix pitch.

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Number counts of γ-ray bursts

STRONG and Klebesadel[1] suggest that most of the 23 γ-ray bursts detected by the Vela satellites are of galactic origin. I question their arguments, which are based on the distribution in galactic latitude and longitude of those bursts for which coordinates have been determined, as well as on the source counts. They admit that the latitude distribution is not significantly galactic. Similarly, the longitude distribution is of low significance. If, *a priori*, the local spiral arm had been predicted to dominate this distribution, then the observations have a chance probability of $\sim1.5\%$. This probability rises above 6% if the observations in any way suggest the correlation. Further, the location of the event of April 27, 1972 (ref. 2) (which was not used in the reported analysis) does not easily fit into the simple local spiral arm hypothesis.

The source counts as presented give the number of events of time-integrated flux density, S, exceeding some level S_0. As S_0 decreases, the source counts flatten out below the three-halves dependence expected from a uniform source distribution. This seems to indicate a scarcity of weak sources, which is consistent with a galactic origin for the bursts. Such a flattening could perhaps also be caused by the first trigger of the event as well as by the method by which the end of the event is determined. The first trigger effects have been partially eliminated by Strong and Klebesadel and it is evident from their figure caption that the second has been noted, although it is not discussed in the text.

I consider this effect in more detail and assume an abrupt rise and gradual fall-off for the burst represented by:

$$I = 0 \qquad\qquad\quad t < 0$$
$$I = Ke^{-t/\tau} \qquad\quad t \geqslant 0$$