vestigators believe that more detailed sample analysis, including single particles, and simulation studies of ion implantation and meteorite impact will throw light on the respective roles of the solar wind and meteorites in bringing about the synthesis of the observed carbon compounds. Thus, in a recent contribution to *Nature Physical Science* (**245**, 3; 1973) Pillinger *et al.* suggest that the chemically detected iron carbide present may have derived by dissolution of carbon in submicroscopic droplets of metallic iron; these Ångstrom sized droplets seem to have been formed from ferrous iron and solar wind implanted hydrogen during the flash heating induced by micrometeorite impacts.

The unusual synthetic reactions which occur during the evolution of lunar soil particles should therefore continue to provide a fascinating challenge in surface chemistry for some time yet. Just how to solve a number of crucial questions remains to be seen. For example—what is the chemical state of the major proportion of the carbon?

From a Correspondent

TROPONIN

A Molecular Latch?

from a Correspondent

MUSCLE contraction is known to involve the sliding of thick filaments past thin filaments. The control of the contraction by Ca^{2+} ions is thought, at least for vertebrate skeletal muscle, to involve changes in the structure of the thin filaments. Each thin filament is an assembly of three proteins—actin, tropomyosin and troponin—and the way in which these proteins interact and are controlled by Ca^{2+} is currently attracting considerable attention.

In the thin filament, long rod-like tropomyosin molecules are thought to be joined end-to-end to follow the two long-pitched strands of the actin structure, each tropomyosin molecule being associated with seven actin subunits. One molecule of the Ca^{2+}-sensitising protein troponin is attached to each tropomyosin molecule. Changes in the X-ray diffraction pattern of muscle on contraction have suggested that the binding of Ca^{2+} ions to troponin causes the string of tropomyosin molecules to move with respect to the actin structure from a position relatively far from the groove between the two actin strands to a position near the groove. One current hypothesis is that sites on the actin subunits are thereby uncovered and can then interact with the cross-bridges of the thick filament. Conversely, the removal of Ca^{2+} ions from troponin causes the opposite movement of tropomyosin so that the actin sites are no longer available and relaxation is possible.

In order to understand how this movement of tropomyosin can occur, the interactions which troponin makes with actin and tropomyosin need to be defined. The problem is further complicated by the fact that the troponin molecule contains three kinds of subunit—troponin-T (molecular weight 44,000 in chicken), troponin-I (mol. wt 23,000) and troponin-C (the calcium-binding subunit with a molecular weight of 19,000). All three subunits are required for relaxation in actin–myosin–tropomyosin systems. Which of these troponin components binds to actin and which to tropomyosin?

For some time, it has been appreciated that the troponin-T subunit can bind to tropomyosin (which is why the subunit is so named). Hitchcock, Huxley and Szent-Györgyi (*J. molec. Biol.*, **80**, 825; 1973) now produce new evidence that even when troponin-T is excluded, a complex of the other two subunits, troponin-I plus troponin-C, can bind quite strongly to actin–tropomyosin provided that Ca^{2+} ions are absent; in the presence of Ca^{2+} ions the binding is poor. Tropomyosin itself, however, cannot bind the complex of troponin-I and troponin-C; this is deduced from the fact that the appearance of tropomyosin paracrystals or the viscosity of tropomyosin in solution is not altered in the presence of these components. Since troponin-C by itself has no affinity for either tropomyosin or actin, the troponin-I subunit must be responsible for the binding to actin. This result fits in well with observations by other groups (notably that of Perry) that troponin-I can inhibit actomyosin ATPase in the absence of tropomyosin.

Hitchcock *et al.* propose as a working hypothesis that each troponin molecule is bound to the thin filament at two sites: one involves a strong Ca^{2+}-independent interaction between the troponin-T subunit and tropomyosin; the other involves a Ca^{2+}-dependent interaction between the troponin-I subunit and actin. Both interactions are required to obtain relaxation. Calcium, by binding to troponin-C, in some way reduces the binding of troponin-I to actin but has no effect on the binding of troponin-T to tropomyosin.

It has always been difficult to imagine how if troponin bound only to tropomyosin it could cause relative movements of actin and tropomyosin on binding Ca^{2+} ions. Now that there is evidence that troponin can also bind to actin in the absence of Ca^{2+} ions, it is possible to speculate how this movement might be brought about. For example, in the absence of Ca^{2+}, the troponin might serve to secure the tropomyosin molecules to the actin structure in the position producing relaxation, whereas in the presence of Ca^{2+} the troponin would no longer bind to actin and the tropomyosin molecules would be free to adopt the position producing contraction. At all events, with five polypeptide components and at least five types of protein-protein contact the thin filament clearly provides an abundance of interactions for the allosterically-minded.

JUVENILE HORMONE

Delayed Effects

from our Insect Physiology Correspondent

ONE of the most promising effects of the juvenile hormone and its synthetic mimics, considered as potential agents for pest control, has been the disruptive action they can exert on subsequent development when applied to the pregnant female or to the developing eggs. In some insects not only can embryonic development be so upset that the eggs fail to hatch, but apparently normal larvae may show signs of excessive juvenile hormone production in the later stages of development: they may give rise to non-viable 'adultoids', or intermediates between larva and adult, at the time when they would have been expected to undergo normal metamorphosis.

This delayed effect was at first attributed to the persistence of traces of the active substance as a contaminant of the egg shells from which the treated insects had emerged. But Riddiford and Truman (*Nature*, **237**, 458; 1972) proved convincingly that in the linden bug *Pyrrhocoris apterus* there is a lasting disturbance in the secretory activity of the corpus allatum in the treated insects. It seems that in such insects the corpus allatum continues to secrete juvenile hormone in the fifth instar and thus to maintain larval characters when the insect should have become adult. Excision of the corpus allatum from such larvae led to normal metamorphosis; transplantation of their corpus allatum into normal fifth instar larvae caused these to transfer into adultoids.

Effects of this sort do not occur in all insects. In the cabbage butterfly *Pieris* there is no such delayed effect on metamorphosis. It has now been shown by Hunt and Shappirio (*J. Insect Physiol.*, **19**, 2129; 1973) that in the lesser milkweed bug *Lygaeus kalmii* the excessive hormone secretion, induced by the application of juvenile hormone analogues to the developing embryo at about the time of blastokinesis, will not only prevent proper metamorphosis of the fifth instar but will lead to the distinctive colour pattern of the early larval instars being retained in the later