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Reply to Criticisms of a Quantum-Mechanical Muscle Model

THE fundamental idea in a highly speculative new kind of muscle model[1] was that a bond vibration is first produced by an exothermic chemical reaction, the energy is transferred resonantly to an electronic excited state of identical frequency, then trapped in an excimer state formed between two such electronic oscillators and finally converted into external work as the two oscillators approach each other. The first part of Naqvi's criticism[2] was that the experiment I quoted as an example of energy trapping in excimers was mistaken. This I concede; clearly the pyrene experiments can no longer be used to show that excimer fluorescence is dipole-forbidden. The accepted theory clearly predicts, however, that, in special conditions, the excimer state may yet be long lived. In its longer version[3] my muscle model already has a design feature which enables Naqvi's criticism to be overcome. If the oscillator was indeed first excited to a singlet state and only later diffused into interaction distance with another oscillator in the ground state then, as Naqvi argues, the triplet state would be at a lower energy and decay into it would be difficult to avoid. My suggestion[3] was, however, that the energy of the bond vibration produced on ATP hydrolysis is less than that of the excited state of an isolated oscillator, so that only when the two oscillators are close enough to perturb each other sufficiently do they resonate with the bond vibration and then become excited. This feature was introduced, first, to protect the postulated bond vibration (by allowing it to be buried in a protein and yet to interact with the environment in this way) and, second, to ensure that only the anti-symmetric attractive excimer state is produced on excitation. I now suggest a third function: to ensure that the excimer only becomes excited when its energy is already below that of the triplet state. In this case decay into the triplet state would be prevented, and the fluorescence would become fully dipole-forbidden.

Naqvi also argues[2] that thermal motions must lower the symmetry of the excimer state, causing it to decay. This conclusion neglects the evolutionary capacity of a protein molecule. All that is necessary is that a protein should have evolved which holds the oscillators rigidly enough parallel and yet allows them to approach each other; such a structure is easy to devise mechanically. Liquid dye lasers protect ions in their excited states from thermal decay by, in some cases, surrounding the ion with chelate cages to prevent random collisions; a protein may well do something similar.

The criticism of Banks, Callomon and Vernon[4] was that the rapidity of vibrational relaxation in free solution makes it physically impossible to store the postulated bond vibration away from thermal equilibrium for the time necessary for the above model. Such relaxation is indeed a crucial difficulty in my muscle model, and one I have considered elsewhere[3]. The process of relaxation[5] takes place on collision and the whole of the vibrational energy has to be converted in one step into a combination of alternative vibrational, translational and rotational modes. Thus the rate of relaxation depends critically on the energy gap between the relaxing vibration and the nearest mode (usually another vibration) beneath it. On this basis the theory of Schwartz, Slawsky and Herzfeld[6] makes predictions which are consistent with experiments[5]. In extreme cases[5], with large energy gaps, a vibration can last for as much as 10 s (N_2) or 6 s (CO), and not merely in dilute gases as Banks, Callomon and Vernon claim but at 1 atm and 0° C. In a liquid, relaxation is faster because of the 100-fold increase in collision rate, but its mechanism is the same. An answer to the problem therefore is to select (or evolve) an environment for a vibrating bond in which translational motions are cut to a minimum, rotations are prevented and neighbouring bonds are far from resonance. I am postulating[1,3] that a special site has evolved in a protein for this function; this is by no means impossible. It is well known[7] that proteins have hydrophobic cores and that enzymes need to hold substrates in such pockets to work at all. The figure of 10^{-7} s I suggested[1,3] for the relaxation time was conservative. Apart from this point, my model depends less on the absolute lifetime of the state than on the relative rapidity of resonant energy transfer ($\sim 10^{-14}$ s). Thus there is no need to invoke new principles in this model, but only to make use of what is already well known in other fields.

Banks, Callomon and Vernon[4] have also criticized my statement of the Second Law of Thermodynamics on the grounds that I was only led to make it because I assumed *a priori* that ATP can store energy. This was not the case; in fact I was led[1,8] to restate the second law to solve the problem raised by Popper[9], namely to find a statement consistent with Brownian motion and thus applicable at the molecular level. This was achieved[8] by defining stored energy and heat relative to the cycle time of the machine which attempts to use these energies. In effect (but not, I think, explicitly) spectroscopists have been doing this for years. Experimentally there are many processes (such as fluorescence, phosphorescence, chemiluminescence and photosynthesis) that trap and store energy in single molecules[10]. I am merely suggesting that ATP stores energy in a similar way.

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