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Quantum optics

Energy transfer under control

William L. Barnes and Piers Andrew

Life on Earth is ultimately sustained by photosynthesis. This phenomenon makes use of perhaps the most fundamental example of a process known as excitation-energy transfer[1]. Solar energy is harvested by 'antenna' molecules that readily absorb photons of light, and is then transferred with great efficiency to a distant reaction site for use in metabolic processes. This transfer mechanism has been widely studied; it has even proved possible to mimic this aspect of photosynthesis in artificial light-harvesting structures based on molecular films[2]. Adopting this energy-transfer process in optoelectronics is an attractive idea that allows us to tailor the characteristics of a new generation of devices, such as polymer light-emitting diodes[3] and lasers[4].

Developments in quantum optics have shown that the interaction of light and matter can be controlled by manipulating the local optical environment[5]. Microcavities and photonic bandgap materials have been used to control spontaneous emission, in which an excited molecule gives up its energy as light. Can the same control be exerted over the energy-transfer process? A report in *Physical Review Letters*[6] by Hopmeier *et al.* indicates that such control may indeed be possible, and could have far-reaching implications.

Excitation-energy transfer takes place between a donor molecule, initially in an excited state, and an acceptor molecule, usually in its ground state (Fig. 1). Provided there is sufficient overlap between the emission spectrum of the donor and the absorption spectrum of the acceptor, energy may be transferred. If the donor and the acceptor are far apart then the transfer is radiative — the donor emits a photon that is subsequently absorbed by the acceptor. For smaller separations, less than the wavelength of the light involved, the dipole moment associated with the excited donor may induce a dipole moment in the acceptor. The resulting dipole–dipole interaction leads to a non-radiative transfer of energy known as Oppenheimer, or Förster[7,8], transfer.

By placing an excited molecule between two closely spaced mirrors to form a microcavity, the process of spontaneous emission may be controlled. The boundary conditions imposed by a microcavity lead to resonant modes when the wavelength fits neatly within the cavity. If emission takes place at this resonant wavelength it will be enhanced. Several recent theoretical studies indicate that the energy-transfer process can be controlled in the same way as spontaneous emission by confining the molecules within, for example, a microcavity[9,10] or a photonic bandgap material[11,12].

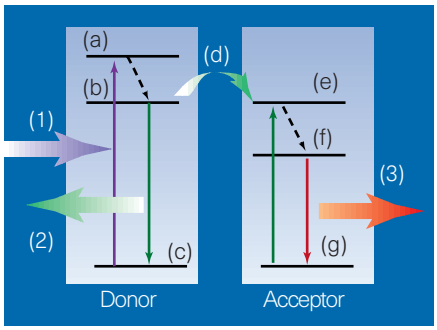

Figure 1 **Energy-transfer scheme. Light** (1) **is absorbed by the donor, promoting it to an excited state** (a) **from which it relaxes non-radiatively to a metastable level** (b)**. From here it may either return to the ground state** (c) **to produce a photon** (2)**, or transfer its energy** (d) **and thus promote the acceptor to an excited state** (e)**. The acceptor then relaxes** (f) **and may subsequently decay to the ground state** (g) **to produce a photon** (3)**. The energy-transfer step** (d) **is the one that Hopmeier *et al.*[6] are now able to control through the optical environment in which they place the donor and acceptor.**

Hopmeier *et al.*[6] have now studied the energy-transfer process in a well-defined microcavity consisting of a dielectric stack mirror on one side and a metallic mirror on the other. They studied energy transfer from a light-emitting polymer to an organic dye confined within this cavity. The polymer (donor) was excited with an ultraviolet laser, and emission from the dye (acceptor) was collected in the red part of the spectrum. By varying the thickness of the mixed polymer/dye layer separating the microcavity mirrors, they were able to sweep the resonance wavelength of the cavity all the way across the spectral region associated with energy transfer from polymer to dye. Using both intensity- and time-resolved measurements, they showed that a significant enhancement in emission from the dye

100 YEARS AGO

"To those who study the progress of exact science, the common spinning-top is a symbol of the labours and the perplexities of men who had successfully threaded the mazes of the planetary motions. The mathematicians of the last century, searching through nature for problems worthy of their analysis, found in the toy of their youth ample occupation for their highest mathematical powers. No illustration of astronomical precession can be devised more perfect that that presented by a properly balanced top, but yet the motion of rotation has intricacies far exceeding those of the theory of precession. Accordingly we find Euler and D'Alembert devoting their talent and their patience to the establishment of the laws of the rotation of solid bodies. Lagrange has incorporated his own analysis of the problem with his general treatment of mechanics; and since his time Poinsot has brought the subject under the power of a more searching analysis than that of the calculus, in which ideas take the place of symbols, and intelligible propositions supersede equations." (Maxwell – "Collected Works", I. p. 248).
From *Nature* 3 August 1899.

50 YEARS AGO

Although much had been written and much had been done, nevertheless neither the Medical Research Council in Great Britain nor the National Research Council in the United States of America was in a position to give any definite answer when, at the beginning of the War, their respective Governments asked for information about the amounts of certain nutrients required by the human body. … The intention was to give a group of volunteers "a diet virtually devoid of vitamin A and carotene until unmistakable signs of deficiency appeared, and then to determine what dose of vitamin A or carotene was needed to ensure recovery to normal". … Perhaps the most unexpected findings in the present instance were the "prolonged delay before the onset of nutritional changes" (eight months of deprivation produced no discernible indication of deficiency). … Defective night-vision, with a raised rod-threshold and prolonged cone–rod transition-time, and a lowering of the plasma level of vitamin A proved to be the first definite signs of deficiency.
From *Nature* 6 August 1949.

occurred when the wavelength of the cavity mode coincided with that of the transfer process.

The authors' samples contained a random distribution of donors and acceptors within the cavity region. As a consequence, both radiative transfer between widely separated donor–acceptor pairs and non-radiative transfer between neighbouring pairs is expected. They argue that the increased acceptor emission they saw resulted from the microcavity enhancement of the radiative energy-transfer process. So, their results strengthen previous findings involving the optical modes of liquid microdroplets[13].

The potential applications of this work are many and varied — for example in the design of organic light-emitting devices — but our knowledge of the underlying physics remains incomplete. The question remains as to whether the non-radiative transfer process is affected in a similar fashion. Further work with well-defined donor–acceptor separations is needed to give a clearer picture of the different transfer mechanisms. Nonetheless, it looks increasingly likely that energy transfer modified in this way will extend our ability to control the interaction between light and matter. One of the most recent uses to which energy transfer has been put is to control the wavelength and efficiency of solid-state semiconducting organic lasers[4]. In the future, modifying the transfer process in the manner suggested by Hopmeier *et al.*[6] should improve the efficiency of these lasers. This approach might also be used to control energy transfer between quantum dots[14], and could perhaps be used to develop efficient artificial photosynthetic devices. □

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Malaria

A blueprint of 'bad air'

Mats Wahlgren and Maria Teresa Bejarano

The 'bad air' agent — *mal aria* in Italian — was fancied to be the cause of malaria far into the nineteenth century. At that time, Ross and Grassi discovered that mosquitoes transmit these microscopic protozoa. Just as this discovery was a big leap then, the next step today is to discern the parasite's blueprint; the genomic sequence of *Plasmodium falciparum*. Four hundred million people still suffer from malaria worldwide, and a million children die each year in Africa alone. There is no vaccine, antimalarial drugs are failing, and many sufferers also develop tumours (such as Burkitt's lymphoma).

On page 532 of this issue, Bowman *et al.*[1] unveil the entire sequence of the *P. falciparum* chromosome 3. These authors have sequenced and analysed a million bases, which, along with the previously sequenced[2] chromosome 2, now gives us 7% of the total *P. falciparum* genome (roughly 30 mega base-pairs, Mb). This complex genome comprises 14 chromosomes, each equivalent in size to a complete bacterial genome (0.65 to 3.4 Mb), as well as mitochondrial- and plastid-like sequences. Some 215 protein-coding genes are found on chromosome 3, compared with 209 on chromosome 2, giving an estimated 6,500 genes in the whole genome.

Perplexing though the *P. falciparum* genome may seem, a certain organization has been found. Clusters of genes encoding proteins produced at similar stages in the parasite's life cycle, or proteins with similar functions, seem to be grouped in particular chromosomes or chromosomal locations. For example, in chromosome 2 there are many genes encoding proteins with high levels of the amino acid serine, and also genes encoding structures found on the surface of the parasite (merozoites)[2]. Another example of gene clustering is the sexual-stage-specific genes, which are localized to chromosome 5 in a related malaria parasite. And a sequence that may be involved in separating a chromosome from its sister copy during cell division has been identified at roughly the centre of chromosomes 2 and 3. Known as the centromere, it consists of 2 kb of repeated adenine–thymine-rich sequences. However, the core of the *P. falciparum* centromere is shorter than those in other eukaryotes, and it remains to be shown whether it functions as a centromere. Such comparative analyses of chromosomes, particularly between different malarial parasites (all of which have 14 chromosomes)[3], should help in understanding genome structure and evolution.

The malaria parasite develops inside red blood cells, where it is protected from most mechanisms of the immune system. But the parasite exports some of its own polypeptides to the surface of the red blood cell (Fig. 1), and here it is open to immune attack. To fool the immune system, it regularly exchanges these polypeptides ('antigenic variation')[4]. The polypeptides are encoded by members of the *var* (variable) and *rif* (repeated-interspersed family) gene families, which are found close to the ends (telomeres) of both chromosomes 2 and 3 — each telomere is flanked by one *var* gene and several members of the *rif* family. So far, nine *rif* genes (including two members of the *stevor* sub-family) have been identified in the subtelomeric regions of chromosome 3, and 17 members (including four stevors) have been found in the same regions of chromosome 2. This gives an estimated 200 *rif* genes per genome, making it the largest gene family in *P. falciparum*. Bowman and colleagues' results[1] make the picture even more complicated, as they have discovered more gene families — the *clag* (cytoadherence-linked asexual gene) and CTP (conserved telomeric protein) families, for example — in the subtelomeric region of chromosome 3.

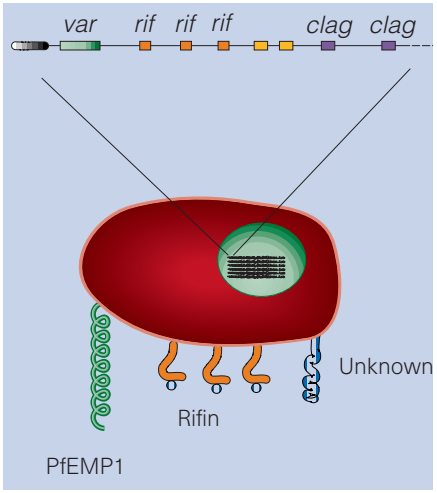

Figure 1 **Red blood cell infected with *Plasmodium falciparum*. Antigens expressed at the surface of the blood cell are shown (bottom), as well as the end of chromosome 3 (top), where Bowman *et al.*[1] have identified the genes that encode these proteins. The antigens are highly variable, particularly those encoded by members of the *var* and *rif* gene families (PfEMP1 and rifin, respectively). There are roughly 50 *var* genes, although only one is expressed at a time[11,12]. By contrast, several of the 200 or so *rif* gene products may be present on the surface of the same red blood cell. The *clag* gene family is smaller, and it is not yet clear whether the proteins encoded by these genes are expressed at the surface of the red blood cell[7]. However, parasites that lack the *clag* gene on chromosome 9 adhere poorly to host cells[10]. (Yellow boxes depict genes encoding proteins of unknown function.)**