PERSPECTIVES: POLYMER PHYSICS

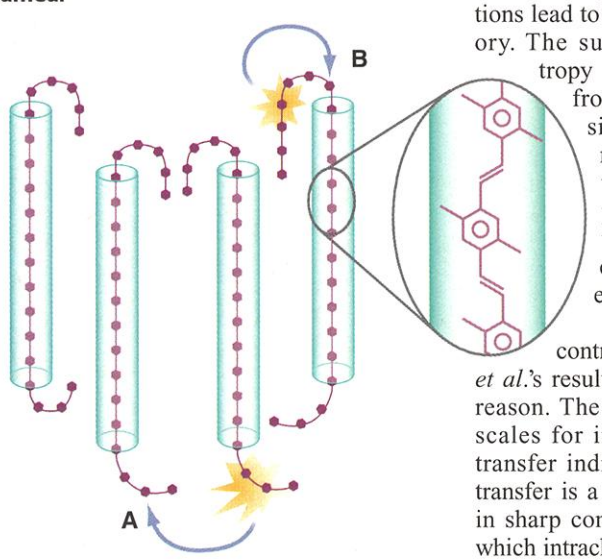
Prospects for the Polymer Nanoengineer

Sumit Mazumdar

The ongoing quest for new materials based on conjugated polymers is driven by two goals: to understand their intrinsic properties and to improve the performance of organic optoelectronic devices. Theoretical and experimental studies of the photophysics of conjugated polymers often assume an infinite defect-free quasi–one-dimensional chain or an ordered chain arrangement with predictable interchain interactions. But the morphologies of conjugated polymer films are much more complex, with twists and bends on individual chains that reduce the effective conjugation lengths considerably from those expected on the basis of molecular weight analyses. Real materials therefore have a distribution of conjugation lengths, which in most cases peaks at a modest conjugation length. The coiled nature of the chains and the cross-linking between them lead to interchain interactions that further modify charge and energy transfer.

To improve the photoluminescence or electroluminescence quantum yields of devices using conjugated polymers, charge and energy transfer must therefore be controlled in the bulk system. This is a formidable task, requiring the design of ingenious architectures with desirable chain ordering and separations by self-assembly and/or nanoengineering. On page 652 of this issue, Nguyen *et al.* (*1*) control energy transfer in a nanoengineered conjugated polymer, poly[2-methoxy-5-(2′-ethyl-hexyloxy)-1,4-phenylene vinylene] (MEH-PPV), by using host-guest chemistry in an oriented mesoporous host (*2*). The mesoporous silica host has aligned pores that are hexagonally ordered. Since their discovery (*3*), these host materials have been used to design a variety of nanoscale materials by incorporating various guest systems into the pores. The materials used by Nguyen *et al.* have pore diameters of 2.2 nm, small enough for a single MEH-PPV chain to be incorporated into each pore. Furthermore, individual chains are isolated from each other by a thick dielectric that precludes energy transport between the chains (*2*).

From polarized photoluminescence measurements and from the distribution of pore angles measured with x-ray diffraction, Wu *et al.* concluded previously that more than 80% of the polymer chains are inside the aligned pores, with the remaining ones staying outside the pores (*2*). The photophysics of the chains inside the pores should mimic the behavior of isolated chains, as confirmed in careful measurements of the dynamics of polarized excited state absorptions by femtosecond pump-probe spectroscopy (*1*). The dynamics of the states reached by excited state absorption of the polymer chains inside the pores follow that of highly excited isolated chains in solution. In contrast, the excited states of the polymer chains outside the pores mirror the behavior of spin-cast thin films.



Energy transfer processes in nanoengineered MEH-PPV. Single polymer chains are incorporated in individual silica pores. Optical excitation polarized parallel to the pores leads initially to rapid energy transfer between chain segments that are outside the pores (**A**). At later times this is followed by relatively slow, directed intrachain migrations of the excitons into the pores (**B**).

Nguyen *et al.* also demonstrate controlled energy transfer by using time-resolved luminescence studies to measure the time-dependent luminescence anisotropy, which is the difference in intensity between light emitted parallel and perpendicular to the silica pores when the excitation laser is polarized along the pores. The anisotropy initially decays in ultrafast time (within about 1 ps), but this is followed by a relatively slow exponential rise (with a time constant of about 250 ps). The initial fast decay of the anisotropy results from interchain energy transfer between the polymer segments outside the pores, whose random orientations lead to the loss of polarization memory. The subsequent growth in anisotropy is attributed to luminescence from the oriented segments inside the pores, indicating directed energy transfer from the segments outside the pores to the segments inside, with the latter having larger conjugation lengths and smaller optical gaps.

In addition to demonstrating controlled energy transfer, Nguyen *et al.*'s results are important for a second reason. The large difference in the time scales for intra- and interchain energy transfer indicates that interchain energy transfer is a much slower process. This is in sharp contrast to carrier transport, for which intrachain mobility is larger than interchain mobility by several orders of magnitude (*4*). This difference between charge and energy transfer on a single chain is in agreement with calculations of model conjugated polymers that incorporate the electron-electron interaction between the π electrons. The lowest optical state within these models is an exciton, an electron-hole pair that, being a composite particle, has a considerably smaller bandwidth than the carrier bandwidth (*5*). Energy transfer due to dipole-dipole coupling is largely ineffective between segments of the same chain with a kink or twist, and a single kink can therefore severely reduce the exciton motion along a chain.

Nguyen *et al.* thus clearly demonstrate isolated chain behavior inside the silica pores and conformation-driven energy transfer. Their study shows that measurements of luminescence polarization memory provide a powerful technique to deduce energy transfer in conjugated polymers. Nguyen *et al.*'s system can be considered a periodic array of molecular wires, and there is currently great interest in molecular electronics and optoelectronics of such systems (*6*). In particular, molecular wires consisting of simple conjugated polymers can perhaps serve as simple models for more complicated systems such as DNA molecules and conductive nanowires (*6*).

Device applications using such nanoengineered polymers will, however, have to overcome additional challenges. Fast intrachain carrier transport and slow energy transfer may have disadvantages in

The author is in the Department of Physics and the Optical Sciences Center, University of Arizona, Tucson, AZ 85721, USA. E-mail: sumit@physics.Arizona.edu

light-emitting diodes, in which a dominant mechanism of exciton quenching involves polaron-exciton collision processes (*7*); these processes will be enhanced in the confined one-dimensional molecular wire. Precise control of polaron and exciton mobilities may overcome this problem in the future. On the positive side, polymers incorporated within the silica nanopores are less susceptible to air oxidation (*2*). From a basic science perspective, various interesting photophysical experiments using the nanoengineered polymers can be envisaged. For example, details of the difference between ultrafast photoinduced absorptions in solutions and in thin films of PPV (*8*, *9*) are yet to be understood. Further studies of the excited state absorptions in the nanoengineered PPV, especially the low-energy excited state absorption in the midinfrared region (*8*, *9*), may elucidate the differences between intra- and interchain processes in conjugated polymers. As clearly illustrated by Nguyen *et al.*, nanoengineered samples hold much promise for elucidating the photophysics of conjugated polymers and designing advanced optoelectronic devices.

References and Notes

1. T. Nguyen, J. Wu, V. Doan, B. J. Schwartz, S. H. Tolbert, *Science* **288**, 652 (2000).
2. J. Wu, A. F. Gross, S. H. Tolbert, *J. Phys. Chem. B* **103**, 2374 (1999).
3. C. T. Kresge *et al.*, *Nature* **359**, 710 (1992).
4. R. J. O. M. Hoofman *et al.*, *Nature* **392**, 54 (1998).
5. The exciton bandwidth is proportional to the square of the carrier bandwidth divided by the exciton binding energy [F. B. Gallagher and S. Mazumdar, *Phys. Rev. B* **56**, 15025 (1997)].
6. See abstracts by J. Barton, E. Braun, H. W. Fink, M. Ratner, and D. Porath, Annual March Meeting of the American Physical Society, Focused Session on DNA and Molecular Conductance [published in *Bull. Am. Phys. Soc.* **45**, 50–51 (2000)].
7. W. Graupner *et al.*, *Phys. Rev. Lett.* **77**, 2033 (1996).
8. M. Yan *et al.*, *Phys. Rev. Lett.* **75**, 1992 (1995).
9. S. V. Frolov *et al.*, *Phys. Rev. Lett.* **78**, 4285 (1997).

PERSPECTIVES: NEUROBIOLOGY

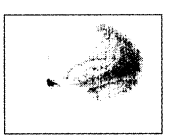
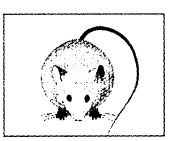
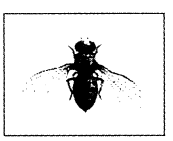
Of Flies and Mice

Valina L. Dawson

Parkinson's disease (PD) was first described 180 years ago yet the cellular mechanisms that cause a select group of dopamine neurons to die, resulting in this common neurodegenerative disorder, are unknown. Identification of mutations in the proteins α-synuclein, parkin, and the ubiquitin hydrolase UCH-L1, in patients with the rare inherited forms of PD, has yielded opportunities to understand the pathogenesis of this insidious disease through modern molecular approaches (*1*, *2*). Animals engineered to express human genes linked to Alzheimer's disease, amyotrophic lateral sclerosis, and Huntington's disease have already provided valuable mechanistic insights into the pathogenesis of these dreaded diseases and are presenting exciting treatment possibilities for patients (*3*). Two recent papers now report on the first PD animal models in the fly and the mouse, which have some but not all of the cardinal features of PD. Masliah *et al.* (*4*) describe transgenic mice that overexpress human wild-type α-synuclein, whereas Feany and Bender (*5*) have engineered transgenic fruit flies to overexpress either wild-type human α-synuclein or a form of the protein that carries the PD mutations A53T (substitution of alanine at position 53 by threonine) or A30P (substitution of alanine at position 30 by proline).

PD is a movement disorder characterized by rigidity, tremor, slowed movement, and impaired gait and in some cases by impaired memory and cognitive dysfunction (*6*). The hallmark pathological feature is the progressive degeneration of dopamine neurons in an area of the brain called the substantia nigra pars compacta. An estimated 60 to 80% of dopamine neurons are lost before characteristic clinical signs of PD become manifest (*7*). Accompanying neuronal loss is the formation of inclusions in the cytoplasm of neuronal cell bodies (Lewy bodies) or in the extensions of neurons (Lewy neurites). These are spherical, dense cytoplasmic inclusions with a pale halo (first described by Lewy in 1912) that contain predominantly α-synuclein but also ubiquitin and other proteins (*6*, *7*). Lewy bodies are found in other neurodegenerative disorders but generally are considered to be a defining pathological feature of PD in conjunction with loss of dopamine neurons (*8*).

The PD model in the fruit fly developed by Feany and Bender (*5*) reproduces many features of the human disease. Expression of the human gene encoding wild-type α-synuclein or transgenes engineered to carry the A53T or A30P mutations results in an age-dependent loss of dopamine neurons in the fly, starting at mid-life. The loss of dopamine neurons is restricted to the nervous system and not all dopamine neurons are lost, reminiscent of the human disease in which dopamine neurons in the substantia nigra die, but those in the ventral tegmental area are spared. Structural analysis reveals neuronal cytoplasmic inclusions that have a dense core with radiating filaments and a halo reminiscent of Lewy bodies in humans. This pathology is not seen in normal aged flies. The flies express a progressive, age-dependent loss of motor function as measured by climbing activity in all three lines of transgenic flies, but loss of climbing activity

	 Human	 Mouse	 Fly
	Age-dependent onset with chronic progression	Age-dependent onset—unknown	Age-dependent onset with chronic progression
		Inclusions get larger with age	
	Dopamine neuronal cell loss in select brain regions	Dopamine neuronal cell injury	Dopamine neuronal cell loss
	Lewy bodies (cytoplasmic inclusions containing α-synuclein and ubiquitin with a core and radiating fibrils)	Cytoplasmic inclusions (containing α-synuclein and some ubiquitin without fibrils); nuclear inclusions	Cytoplasmic inclusions (containing α-synuclein with fibrils; ubiquitin not determined)
	Motor deficits	Motor deficits	Motor deficits
	Mitochondrial complex 1 deficits	Unknown	Unknown
	Increased markers of oxidative stress	Unknown	Unknown

A comparison of animal models of PD. Recent molecular advances have enabled the engineering of mice and flies that carry wild-type or mutant versions of the protein α-synuclein, which is implicated in PD. A comparison of the features of the fly and mouse animal models of PD and how they correlate with the characteristics of the disease in human patients is shown.

The author is in the Department of Neurology, Johns Hopkins University School of Medicine, 600 North Wolfe Street/Carnegie 214, Baltimore, MD 21287, USA. E-mail: vdawson@jhmi.edu