POLYMER SCIENCE

Capturing the Dynamic Behavior of Adsorbed Polymers

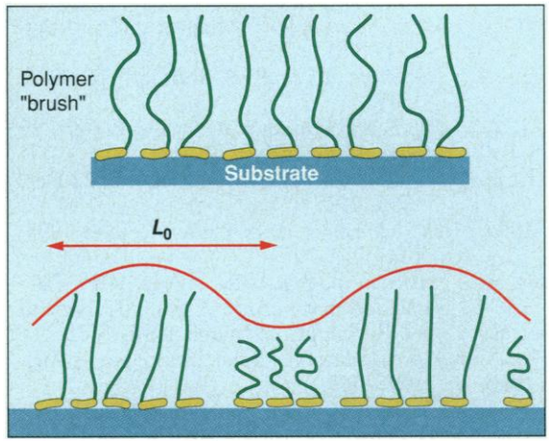
Anna C. Balazs

An adsorbed layer of polymer can dramatically alter the properties of a surface. A coating of polymer on a relatively inert surface, for example, can enhance the adsorption and adhesion of a subsequent layer. Such "primers" act as a glue between the substrate and outer coating and facilitate the uniform spreading of paints or protective films. Adsorbed polymers can also be used to hinder surface adsorption. For instance, a coating of hydrophilic chains can inhibit the deposition of proteins and platelets and thereby enhance the biocompatibility of artificial implants (*1*). Polymers adsorbed to the surface of the influenza virus prevent its binding to the surface of red blood cells and consequently inhibit the spread of the disease (*2*). Spraying a polymer layer on industrial molding machines prevents extensive sticking and ensures the quick release of injection molded parts, such as car bumpers.

The ability of the chains to display such interfacial activity is dependent not only on their static structure but also on their dynamic behavior. Until now, our knowledge of the adsorbed chains was confined to their static conformations. On page 2041 of this issue, however, Fytas *et al.* report on a technique that allows measurement of the dynamic fluctuations within the layer (*3*). This method now makes it possible to investigate how the collective microscopic motion of polymers at interfaces affects such macroscopic phenomena as adhesion, lubrication, and wetting.

Fytas *et al.* focus on the properties of terminally anchored polymers (*3*). The polymers are composed of two distinct blocks: a long polystyrene block and a shorter polyethyleneoxide fragment. The chains and substrate are immersed in a solvent that is compatible with the polystyrene but incompatible with the polyethyleneoxide fragment. The solvent incompatibility drives the polyethyleneoxide to bind to the surface and thereby anchor the chains to the substrate. The polystyrene blocks stretch into the favorable solvent, forming "bristles" (figure, top); the layer is referred to as a polymer brush (*4*).

The height of the brush is dependent on the molecular weight of the solvophilic block, and thus, the chains can form a relatively thick layer. Thick films play an important role in a number of technologically relevant applications. When long chains are terminally anchored to the surface of colloidal particles, the steric repulsion between the tethered layers prevents the particles from aggregating and precipitating out of solution (*5*). It is through this mechanism, for example, that dye and pigment particles stay suspended in solution. The thick layers also form effective lubricants, reducing the friction and wear between mechanical parts. Finally, these tethered and extended macromolecules act as model systems for the chains within lipid bilayers; thus, investigating the interactions between the layer and other chains or the surrounding solvent provides insight into the properties of the bilayer.



Doing the polymer wave. (**Top**) Terminally anchored chains form brushlike structures on the surface. Polystyrene blocks are green, and polyethyleneoxide fragments are yellow. (**Bottom**) The deformations in the anchored layer. Some chains are compressed and others are stretched. The size of the deformations is comparable to $L_0 \sim 2\pi/q$, where L_0 is the layer thickness and q is the difference in scattering wave vectors.

Because of the potential utility of the these thick, end-grafted layers, considerable attention has been focused on determining their properties. The majority of the earlier studies have focused on characterizing the static equilibrium properties of the layer. For example, both experimental measurements (*6*) and theoretical calculations (*4*, *7*) have been used to probe the density profiles of the layer. Little, however, is known about the dynamic behavior of the brush at thermal equilibrium. Here, thermal fluctuations (thermally activated collective motions of the chains) cause part of the chains to be more extended and other regions to be more compressed. One could view these motions as undulations in the surface of the brush (figure, bottom). Theoretically, these undulations or deformations are predicted to have a characteristic size, which is comparable to the thickness of the adsorbed layer, L_0 (*8*). Experiments, however, have lagged behind theory in the ability to probe the dynamic behavior of the anchored layer.

Fytas *et al.* used dynamic light scattering to determine the behavior of end-grafted chains (*3*). The advantage of dynamic light scattering is that one can obtain correlations between deformations at times t_1 and t_2. Thus, one can probe not only the characteristic size but also the lifetime of the undulations. In their elegant experiment, a laser beam was aimed at a glass prism that was coated on the opposite side with a layer of end-anchored polymers. The light was totally reflected from the surface of the glass, yet a damped, oscillating electric field from the laser beam penetrated the layer and scattered from the grafted chains. The scattered field characterizes density fluctuations of a size $2\pi/q$, where $q = |\mathbf{q}|$, $\mathbf{q} = \mathbf{k}_s - \mathbf{k}_i$, and $\mathbf{k}_s$ and $\mathbf{k}_i$ are the scattered and incident light wave vectors, respectively. Using this method, the researchers indeed found that the long-lived deformations have a specific size and that fluctuations at other length scales rapidly die away. The results also reveal that the scattering intensity is proportional to $(qL_0)^2$ for small q. Because q is inversely proportional to wavelength, this relation indicates that the long-wavelength disturbances are suppressed.

The findings are significant in that they provide insight into the collective motions of the chains at thermal equilibrium. The technique can now be used to determine how characteristics, such as chain rigidity and solvent quality, affect the deformations of the layer. Thus, the technique can be used as a probe of the local environment. With small modifications to the experimental setup, the method can also be used to determine how the fluctuations are affected by the presence of a second end-grafted layer where the

The author is in the Department of Chemical and Petroleum Engineering, University of Pittsburgh, Pittsburgh, PA 15261, USA. E-mail: balazs@vms.cis.pitt.edu

bristles of both brushes are facing each other. If the separation between the brushes is varied, the results will reveal how the dynamic behavior of lubricating layers is affected as the layers come into contact. The results can also show how the chain deformations can promote or hinder adhesion between the layers. Performing such experiments on biopolymers or lipids can provide information on the fluctuations of the chains within cell membranes.

References

1. R. S. Ward, *IEEE Eng. Med. Biol. Mag.* **6**, 22 (1989).
2. G. B. Sigal, M. Mammen, G. Dahman, G. M. Whitesides, *J. Am. Chem. Soc.* **118**, 3789 (1996).
3. G. Fytas *et al.*, *Science* **274**, 2041 (1996).
4. S. T. Milner, *ibid.* **251**, 905 (1991).
5. D. H. Napper, *Polymer Stabilization of Colloidal Dispersion* (Academic Press, London, 1983).
6. See reference (*7*) in Fytas *et al.* (*3*).
7. A. M. Skvortsov *et al.*, *Polym. Sci. USSR* **30**, 1706 (1988); C. Yeung, A. C. Balazs, D. Jasnow, *Macromolecules* **26**, 1914 (1993); K. Huang and A. C. Balazs, *ibid.*, p. 4736.
8. F. J. Solis and G. Pickett, *Macromolecules* **28**, 4307 (1995).

IMMUNOLOGY

Complexities in the Treatment of Autoimmune Disease

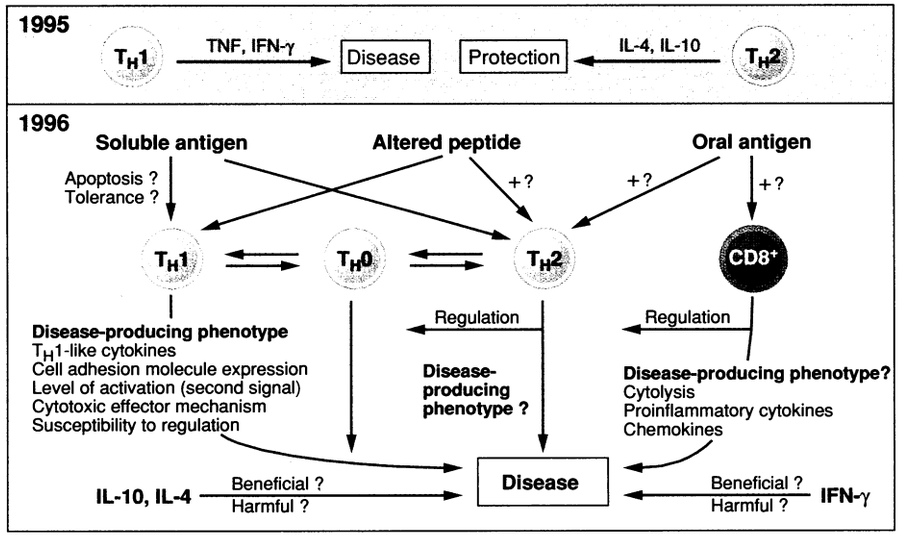
Henry F. McFarland

Two reports, one in this issue (*1*) and another in the 6 December issue (*2*), analyze a promising strategy for treatment of autoimmune diseases—administration of the offending antigen to induce tolerance. The results of both studies are troublesome—they imply that it may be problematic to apply these strategies to humans. In the report in this issue, Genain *et al.* (page 2054) (*1*) show that experimental allergic encephalomyelitis (EAE) induced in marmosets by immunization with myelin oligodendrocyte glycoprotein (MOG) can be delayed by intraperitoneal treatment with soluble MOG but that the treated animals subsequently develop a hyperacute form of disease. In the other report, Blanas *et al.* (page 1707) (*2*) noted that oral administration of large amounts of the antigen ovalbumin (OVA) may result in the generation of $CD8^+$ cytotoxic T cells and that these T cells can increase—rather than prevent—the disease process after oral administration of OVA in a murine model of insulin-dependent diabetes mellitus (IDDM). (In this model, animals are transfected with the gene for OVA on an insulin promoter to allow expression in islet cells and with an enriched population of OVA-specific $CD8^+$ T cells.) Thus, two therapeutic approaches, parental and oral administration of antigen, shown to be successful in mice and now being tried in clinical trials, may under some conditions enhance disease.

Experimental models for IDDM (the NOD mouse) and multiple sclerosis (EAE) have, in the past decade, yielded exciting advances in our understanding of the immunological mechanisms underlying experimental autoimmune disease (see figure). Both IDDM and EAE can be induced by $CD4^+$ T cells, primarily by one of the two types of helper T cell—T helper cells type 1 (T_H1), which produce proinflammatory cytokines such as interferon-γ (IFN-γ) or tumor necrosis factor–α (TNF-α). In contrast, administration or up-regulation of the T_H2-associated cytokines interleukin-4 (IL-4) and IL-10 is beneficial and can ameliorate autoimmune disease. Consequently, current concepts of autoimmune diseases focus on a disease-mediating effect of T_H1-like T cells and a protective effect of T_H2-like T cells (*3*). On the basis of this hypothesis, extensive efforts have been made to identify therapeutic strategies that tilt the T cell response toward a T_H2 phenotype. Many of these strategies—including oral administration of antigen, administration of soluble antigen without adjuvant, administration of modified antigenic peptides, or immune deviation by T_H2-associated cytokines—work in mouse models and are currently in various stages of clinical testing for several autoimmune diseases. Now several recent studies, including the new report by Genain *et al.* (*1*), suggest that the distinction between disease-producing T_H1 T cells and protective T_H2 T cells is overly simplistic and that the relation of proinflammatory and T_H2-associated cytokines in autoimmune disease is extremely complex. For example IL-10 contributes to, or increases, disease in the NOD model (*4*). IL-4, the prototypic T_H2-associated cytokine, can augment autoimmune uveitis in rats (*5*), and T_H2 T cells failed to suppress IDDM in the NOD mouse (*6*). Furthermore, the study by Blanas *et al.* (*2*) raises the possibility that in addition to $CD4^+$ T_H1 T cells, $CD8^+$ T cells may also contribute significantly to some autoimmune diseases. These findings contrast with previous suggestions that oral administration of antigen induces a population of $CD8^+$ T cells that suppress disease (*7*).

The observations in the two new reports must be viewed with some caution. Genain *et al.* speculate that the hyperacute disease was due to the induction of T_H2-like T cells, resulting in an increased antibody response to the antigen. And in fact previous studies in rodents and later in the marmoset have shown that antibody to MOG, which is on the surface of myelin, can enhance demyelination and increase disease (*8*).

However, a number of questions remain unresolved. For example, the authors speculate that residual T_H1-like T cells were responsible for lesion initiation. Could the T_H2-like



Growing complexity.

The author is in the Neuroimmunology Branch of the National Institutes of Health, Bethesda, MD 20892, USA. E-mail: henrymcf@helix.nih.gov