could not be detected in the disordered sample. Three-photon absorption, plotted in Fig. 2, was also modified in agreement with a two parabolic band scaling law,[7] which predicts both scaling and detuning of the coefficient α_3. Finally, A was measured to be around 1 for a TE pump and 0.6 for a TM pump in both set of samples, as shown in Fig. 3.

In conclusion, we have characterized the nonlinear optical properties of a MQW sample before and after intermixing. This process opens up a number of exciting possibilities for integrated all-optical devices.

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CTuQ5 **3:45 pm**

Quantum-confined stark effect in partially strained quantum wells

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In a uniformly strained quantum well (QW), the valence band potential profiles for the heavy-hole (HH) and the light-hole (LH) are square wells with the equal width. Because the HH has a larger effective mass along axis of the growth direction, it experiences a stronger quantum-confined Stark effect (QCSE). This property is undesirable in optical communication applications where polarization-preserving optical fiber is not used. In this paper, we present a novel partially strained QW in which the rate of the QCSE can be separately engineered. We show experimentally and theoretically that in certain cases the HH QCSE is enhanced, while in other cases the LH QCSE is enhanced. It is also possible to achieve bias independent HH and LH degeneracy.

We have inserted thin layers of highly strained GaAs in an unstrained InGaAs QW grown on InP. The thin GaAs layer is under 3.8% tensile strain. The top of the valence band in this layer is split with the LH edge 247 meV above the HH edge. The strained GaAs LH band edge is near the LH band edge of the unstrained InGaAs well material. The LH wavefunction extends throughout the whole QW region with an unchanged effective well width. Conversely, the strained GaAs HH band edge forms potential barriers, which confine the HH. To a first-order approximation, the QCSE is proportional to the effective mass in the growth direction and to the fourth power of the well width. Different effective widths for the HH and LH result in a different QCSE.

We have performed linearly polarized photoluminescence and linearly polarized electroabsorption measurements in total internal reflection mode with the highly strained layers positioned at various locations within the QW. When the thin GaAs layer is grown at the center of the QW, the HH QCSE is enhanced, particularly at lower field. This case is important when polarization-selective modulation is desired. In another extreme, when two GaAs layers are grown at the edge of the QW, the LH moves faster than the HH and thus the QSCE is reversed, a case which may be used to compensate for undesired polarization dependence in regular structures. In a third case, when the GaAs layers are positioned at proper positions, the HH and LH show a bias independent degeneracy that is important for polarization independent light modulator applications.

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CTuR 2:30 pm–4:00 pm
Room 106

Microscopic Bioimaging

Daniel L. Farkas, *Carnegie Mellon University, Presider*

CTuR1 **2:30 pm**

Characterization of photodamage induced by optical tweezers

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Optical tweezers or optical traps are a unique tool capable of manipulating and controlling biological molecules *in vitro* and *in vivo*.[1] A major drawback to using optical traps is the potential for damage produced by the trapping laser in the sample. Optical damage limits the exposure for any trapped specimen and has proved to be a significant problem for optical traps used *in vivo*. Despite these obvious problems, little work has been carried out to characterize or quantify damage produced in optical traps. This has been due, in part, to the unavailability of a convenient biological system to assess the damage.

In this paper we describe a novel biological assay employed in conjunction with an optical tweezers set up to systematically study damage produced in optical traps. The assay is based on tethering cells of *Escherichia coli* (strain KF95[2]) to a glass coverslip by a single flagellar filament. Such cells rotate at speeds that are proportional to their transmembrane potential (protonmotive force), thus providing a direct and quantitative measure of their state of metabolic health.[3] Tethered cells are held by the optical trap and periodically released to monitor their rotation frequency. The rotation rate is thereby established as a function of time in the trap (Fig. 1). The ability to monitor continuously the metabolic health of the cell allows us to observe how the damage occurs as a function of time and to quantify this optical damage. This procedure is carried out for many wavelengths to establish the wavelength dependence of optical damage (Fig. 2). The wavelength dependence shows a damage minimum at 830 nm, where cells live much longer than at 1064 nm, the later wavelength being most commonly used for optical traps. The spectrum of the wavelength dependence is poorly understood, although it is in rough agreement with similar spectra obtained with other biological systems.[4] In addition to the wavelength dependence studies, we have varied the power delivered to the specimen plane to establish the intensity dependence of optical damage. Our optical tweezers (Fig. 3) may be based on any one of three lasers: (1) a tunable Ti:sapphire ring laser (790–1100 nm), (2) a fixed wavelength diode laser (MOPA 991 nm), and (3) a lamp-pumped Nd:YAG laser (1064 nm). These sources allow us to cover a large portion of the near-infrared range at up 200 mW in the specimen plane. On a more practical level, we can simultaneously establish if a wavelength that produces acceptable levels of damage can be identified within the range of an economical, commercial source (a single mode diode laser, for example).

We have demonstrated a biological assay that affords quantitative, real-time and direct assessment of damage in optical traps. Using this assay in conjunction with a broadly tunable optical trap we have established the wavelength dependence of optical damage in a relevant portion of the near-infrared spectrum. In addition, the intensity dependence of photodamage and a quantitative assessment of this damage is described.

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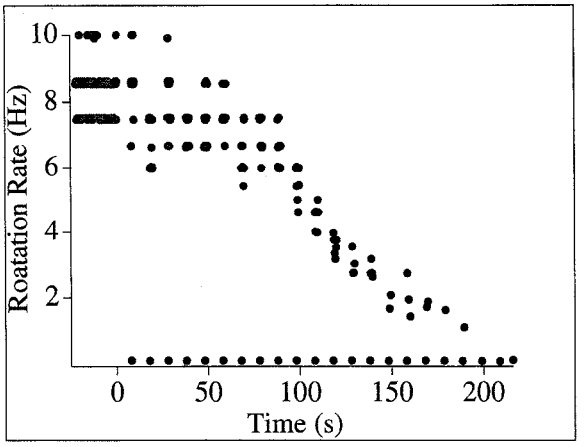
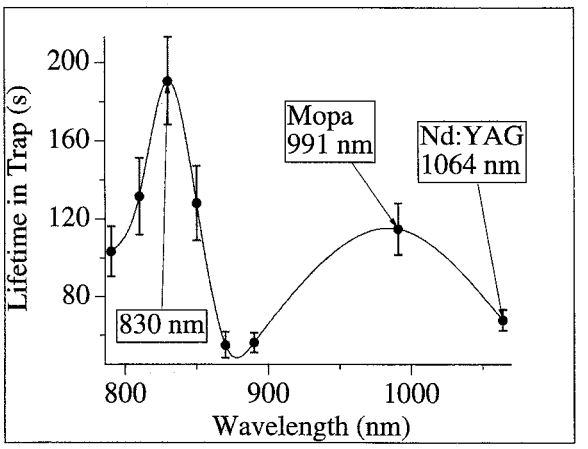
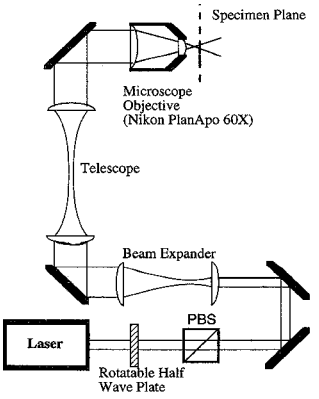


CTuR1 Fig. 1. Raw data showing rotation rate (proportional to the metabolic health) of a single tethered *E. coli* cell as a function of time in the optical trap. The trap is turned on at time zero. The trapping wavelength is 1064 nm and the power in the specimen plane is 50 mW. The very low constant frequency results from the periodic trapping and releasing of the cell. The tendency of the rotation rate to lie on layer lines reflects the 60-Hz data acquisition rate from videotape.



CTuR1 Fig. 2. Lifetime of trapped cells as a function of wavelength. Lifetime is operationally defined to be the time at which a cell's rotation rate decreases to 50% of the initial rate. The power in the specimen plane at each wavelength is set to 100 mW. The first six points (790–890) were generated using the Ti:sapphire laser. The last two were generated with a MOPA diode laser (991 nm) and a Nd:YAG laser (1064 nm).



CTuR1 Fig. 3. The optical tweezers block diagram. The laser sources are: tunable Ti:sapphire ring laser (790–890 nm), single-frequency diode MOPA laser (991 nm), and a single-frequency Nd:YAG laser (1064 nm). The rotatable half waveplate in conjunction with the polarizing beamsplitter (PBS) provides control of the laser intensity. The beam expander allows precise matching of each of the three lasers to the microscope objective entrance pupil. The microscope objective focuses the laser beam, creating the trap, and is also used to image the trapped specimen.

CTuR2 **2:45 pm**

Optical tweezers-based immunosensor

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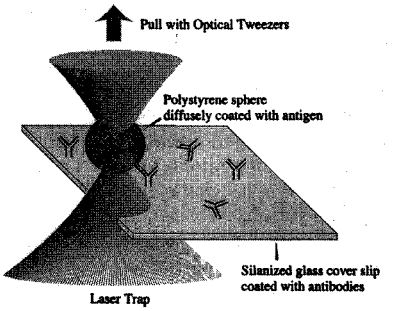
We have developed an extremely sensitive immunoassay that uses optical tweezers to detect antigen-antibody bonds. As shown in Fig. 1, an optical tweezers is used to manipulate a microscopic object with respect to a surface. The adjustable force applied by the tweezers is used to sense adhesion between the objects, which can either naturally or artificially present binding partners. This measurement can be used either to directly detect small amounts of ligand on a surface or to detect low concentrations of free ligand in solution via competitive-binding inhibition of the adhesion.

To develop this immunosensor, we have studied a cell-free system consisting of bovine serum albumin (BSA), covalently coupled to 4.5-μm-diameter spheres, interacting with a surface. In the first experiments, the sensor was used to compare the adhesion force between a sphere and surfaces with and without an anti-BSA coating. The sensor is able to detect bio-specific adhesion due to only a few antigen-antibody binding pairs, even in the presence of nonspecific binding forces.



CTuR2 Fig. 1. Immunosensor measurement technique. In an optical tweezers configuration, the minimum force (i.e., laser intensity) necessary to pull the sphere free of the cover slip can be recorded, allowing either direct measurements of ligand surface density or measurement of free ligand concentrations via competitive-binding effects.

To demonstrate the detection of free ligands in solution, the binding force between a surface with anti-BSA and a BSA-coated sphere was measured as a function of the concentration of free BSA in solution. The free BSA competes with the sphere-bound BSA for binding sites, thereby lowering the adhesion force between spheres and the surface. For concentrations above a picomol/L, the binding force was indistinguishable from that to a surface without anti-BSA, indicating complete displacement of sphere-bound BSA by free BSA. Between a picomol/L and a femtomol/L, the method was sensitive to changes in the binding forces, with the force at a femtomol/L still distinguishable from the force with no BSA in solution. One interesting explanation for the high sensitivity demonstrated for this method may be that the laser light used to manipulate the spheres also attracts free BSA, increasing the local concentration and making it easier to detect.

We have recently begun experiments using this sensor to attempt detection of novel hormone receptors thought to be present on mammary cells. Both *in vivo* and *in vitro*, the human chorionic gonadotropin (hCG) hormone has been shown to promote resistance to the development of mammary tumors and to inhibit the growth of pre-existing mammary tumors in rats.[1] These observations, and the possible application to treatment of human mammary cancer, has led to intense interest in verifying the presence of hCG receptors on mammary cells, which are presumably necessary to mediate the observed interactions. These receptors have thus far eluded detection, but our new immunosensor technique may be able to detect their presence.

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