may therefore provide organisms with a method of varying materials properties while avoiding critical oscillations.

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An Active Gel based on DNA and DNA-Associated Motor Proteins

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Biological systems continuously acquire and use energy sources to perform various functions. This energy is, in part, transduced to generate the forces that control the mechanical behavior of the cell (e.g. cell shape and motion). In this case, the system is in the non-equilibrium state and the material may be called ''Active Soft Matter''.

To investigate the mechanical properties of soft, active systems, we have synthesized an active gel with a well-known semi-flexible biopolymer, DNA, and DNA-associated motor proteins. We study the mechanics of this system using two kinds of microrheological techniques. First, we use a passive measurement in which the intrinsic fluctuation of embedded particles gives information on gel mechanics. Second, we use an active measurement utilizing the forced oscillating motion of embedded particles by an external magnetic field. We discuss these results in comparison to cytoskeletal systems, and seek to establish universal principles of motor-driven active gels.

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Designing Microdevices Operated through Self-Organizations of Microtubules and Kinesin Motors

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Fish melanocytes change their colors through aggregation and dispersion of melanophores. The aggregation and dispersion of melanophores make the appearances of fish melanocytes bright and dark, respectively. The movements of malanophores are driven by biological molecular motors, motor proteins. Inspired by this mechanism, we have envisioned an optical microdevice powered by motor proteins. That is, in arrays of microscale chambers, melanophore-imitated particles, whose surfaces are covered with kinesin motors, are aggregated and dispersed through formations and disassemblies of microtubule asters, respectively. In order to realize such optical device, exploring possible designs of the device is required to test the feasibility of the device. However, laborious experimental procedures hamper such explorations. An alternative way of the exploring would be use of computer simulations. Previously, we have shown the power of computer simulations in designing Lab-on-a-Chip devices powered by motor proteins. Here, we performed systematic explorations of designs of the envisioned device. To this end, we modeled a microtubule as the Kramers chain of a linear polymer, and performed Brownian dynamics simulations. The simulations showed aster formations of microtubules in chambers of various shapes, such as thin triangle, square and hexagonal prisms. We will discuss effects on the aster formations of the size of microscopic chambers, microtubule properties, and motor protein properties. Through the computer simulation, we will show design guidelines for the optical device.

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Concurrent Transcript and Protein Quantification in a Massive Single Cell Array Enables Population-Wide Observation of Oncogene Escape

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The cancer stem cell (CSC) model focuses on the key role these cells can play in drug resistance, since CSCs are believed to reconstitute the cancer after intense chemotherapy treatment. In order to effectively identify and profile CSCs within a heterogeneous tumor population, we are investigating quantitative correlation of messenger RNAs and their translated proteins as a distinctive parameter of the CSC population. However, previous research on mRNA expression and protein abundance has shown the correlations between the two are weak or only ''stochastically meaningful'' due to the significant level of experimental error originating from ensemble observations. Here, we demonstrate a robust microwell-based method to minimize these errors by monitoring both mRNA expression and the corresponding protein abundance from individual cancer cells. Simultaneous observation of membrane protein expression by immunostaining and detection of mRNA transcripts directly from individual cells using a one-step, reverse transcription polymerase chain reaction have been integrated in a massive single-cell array platform. The proposed experimental scheme was initially tested and validated in three established lung cancer cell lines by correlating mRNA transcript and protein expression levels of individual cells and quantitation of heterogeneity. Responses at the individual cell level to known transcriptional and translational inhibitors, as well as to EGFR-specific inhibitors were evaluated, providing quantitative measures of the heterogeneous response of non-small cell lung cancer cells to the inhibitors. Results showed that drug-treated cell lines displayed oncogene escape due to expunction of drug-sensitive subpopulations in the cell lines. Furthermore, correlation of c-MET mRNA and protein levels revealed unique response patterns in different EGFR-mutated cell lines. Thus, these results demonstrate the potential for molecular profiling at the single cell level to prospectively identify the CSCs subpopulation for effective combinatorial treatments.

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Enhancing Automatic Mass Detection in Ultrasound Breast Images using Computer Assisted Detection

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This work is concentrated on extraction of mass in Ultrasound breast images to help radiologists interpreting such images efficiently using Computer-Assisted Detection. A set of six popular ultrasound machines were selected and images were acquired sweeping: modes of operation, transducer, frequency and contrast. To make a complete set of ultrasound images in B-Mode a multi purpose multi tissue Ultrasound Phantom was used. Gamma corrections, contrast stretching and filtering accompanied by morphological Image Processing were among the steps that were applied to find the final image. Two experienced radiologists were marked output. Statistical analysis showed a sensitivity of 100% and accuracy of 99% for solid mass and 99% and 98% for cystic mass respectively. It also showed that the same procedure can be use for cystic and solid breast masses with small changes.

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Modulation of the Invasive Phenotype of Engineered Breast Tumors by the Physical and Cellular Microenvironment

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Tumor development alters the normal cellular processes that maintain tissue integrity and homeostasis, and introduces changes to the tissue surrounding the tumor as well as the tumor cells themselves. Tumor development and invasion are regulated by the physical and chemical properties of the interstitial microenvironment. Here, we examined the effects of both interstitial fluid pressure and vascular endothelial cells on the invasive phenotype of engineered three-dimensional (3D) aggregates of MDA-MB-231 human breast cancer cells. The directionality of the interstitial pressure profile and the presence of endothelial cells altered the frequency at which cells invaded from the surface of the aggregate. Moreover, introducing pressure at one end of an aggregate suppressed invasion at the opposite end. We found previously that elevated interstitial pressure inhibits invasion by altering the chemical composition of the interstitial fluid near the surface of the aggregate. Our data reveal a link between hydrostatic pressure, the vascular endothelium, interstitial convection, and invasion.

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Fibrillar Collagen is Equivalent to Stiff Matrix in Driving Marrow Stromal Cell Differentiation into a Matrix-Deficient, Myofibroblastic-Like Phenotype

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Scars tend to be stiffer than normal tissue, which has prompted the use of stiff matrices as models of scars, but scars are also rich in fibrillar collagen-I. Here, we introduce a soft matrix embedded with distinctly fibrillar collagen type-I, and show that this is sufficient to drive bone marrow stromal cells (MSCs) into a contractile, myofibroblastic-like phenotype – 'myo-MSCs'. These cells have been reported to minimize scarring in a unique wound healing response, exemplified by their application to myocardial infarcts. Transcriptome analysis in response to matrix rigidity points to an upregulation of genes that participate in the cellular contractile machinery, notably α-smooth muscle actin (SMA), but a decreased expression of matrix protein genes for collagen types I and VI, and tenascin-C; TGFβ1 and TGFβRII, implicated in progressive fibrosis,