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Cancer Evolution under Drug-Induced Stress-Gradients[1] GUILLAUME LAMBERT, ROBERT H. AUSTIN, Princeton University — The lack of long term success in eliminating cancer cells while avoiding the evolution of drug resistance indicates that our understanding of how cells evolve in response to stress is still incomplete. We interpret this not as a failure of the current approaches, but rather as an indication that new research venues should be undertaken, where conventional wisdom is challenged in order to drive forward our understanding of cancer. Of particular importance, we believe that the powerful role of evolution in the origin of drug resistance is ill-understood. We do not ask whether evolution occurs, but rather how. We do not describe molecular mechanisms underlying drug resistance at the single cell level, but rather ask how does resistance spread in cancerous tissues and metastatic lesions. We attempt to answer these questions by studying the population-wide dynamics of drug evolution and the collective stress response of cancer cells in a microfluidics device. We use microfluidics technologies to impose high levels of stress on cancer cell metapopulation by create smoothly varying gradients of either oxygen, chemotherapeutic drug, nutrient or pH. We present long-term studies of the adaptation of tumorigenic cancer cells to drug- induced stress gradients.

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