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Using DNA Devices to Track Anticancer Drug Activity DIMITHREE KAHANDA, JASON D. SLINKER, MARC A. MCWILLIAMS, Department of Physics, The University of Texas at Dallas, 800 W. Campbell Rd., PHY 36, Richardson, TX 75080, GAURAB CHAKRABARTI, DAVID A. BOOTHMAN, Departments of Pharmacology, University of Texas Southwestern Medical Center, ND2.210K 601 Forest Park Drive, Dallas, TX 75390-8807, SLINKER LAB COL-LABORATION, BOOTHMAN LAB COLLABORATION — h -abstract-\pard It is beneficial to develop systems that represent significant complexity of biological systems, while maintaining control over specific factors involved in a particular process. We have established a chip-based electrochemical platform for following the repair of DNA damage produced by a redox-cycling anticancer drug, beta-lapachone (β lap). These chips, which possess key features to reproduce the cellular environment, drug cofactors, and base-excision repair (BER) enzymes tracked DNA damage repair activity with redox probe-modified DNA monolayers on gold. The concentration dependence of β -lap revealed significant square wave signal changes at levels of high clinical significance as well as sensitivity to sub-lethal levels of β -lap. We also demonstrate high correlation of this change with the specific drug cycle through rational controls. Thus, this chip-based platform enabled tracking of drug-induced damage repair processes when biological criteria were met, providing a unique synthetic platform for uncovering activity normally confined to inside cells.\pard-/abstract-\

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