1145-60-2995 **Julia L Pelesko*** (jlp192@case.edu), 221 Planet RD, Newark, DE 19711, and **Michael Hinczewski** and **Jacob Scott**. *Curing cancer faster: Optimizing drug scheduling protocols with a Fokker-Plank model.*

Mustonen and Lässig propose a Fokker-Planck equation whose solutions predict how allele frequencies change in populations exhibiting adaptive evolution. In particular, the emergence of resistance to chemotherapy drugs is an adaptive process. Thus, a Fokker-Planck model can be used to predict how populations of cancer cells will evolve under the influence of anti-cancer drugs. Evolutionary steering models already exist to predict drug schedules that minimize the probability of patient relapse; however, current models assume that each drug is taken for infinite time. This assumption is made to ensure genotype frequencies equilibrate before application of a second-line drug. We propose a modified Fokker-Plank model that yields counter-diabatic solutions, or solutions that achieve evolutionary equilibria on finite time scales. Using our model, we can design clinically feasible drug scheduling protocols that minimize the emergence of drug resistant populations of cancer cells. To test our predictions in vitro, we have constructed a morbidostat: an automated continuous culture device. Feedback from the morbidostat will allow us to fine tune the parameters of our Fokker Plank model, resulting in continual optimization of drug schedules. (Received September 26, 2018)