Modeling cell-cycle synchronization during embryogenesis in *Xenopus laevis* R. SCOTT MCISAAC, Princeton University, K.C. HUANG, Stanford University, ANIRVAN SENGUPTA, Rutgers University, NED WINGREEN, Princeton University — A widely conserved aspect of embryogenesis is the ability to synchronize nuclear divisions post-fertilization. How is synchronization achieved? Given a typical protein diffusion constant of $10 \text{µm}^2 \text{sec}^{-1}$, and an embryo length of $\approx 1 \text{mm}$, it would take diffusion many hours to propagate a signal across the embryo. Therefore, synchrony cannot be attained by diffusion alone. We hypothesize that known autocatalytic reactions of cell-cycle components make the embryo an “active medium” in which waves propagate much faster than diffusion, enforcing synchrony. We report on robust spatial synchronization of components of the core cell cycle circuit based on a mathematical model previously determined by in vitro experiments. In vivo, synchronized divisions are preceded by a rapid calcium wave that sweeps across the embryo. Experimental evidence supports the hypothesis that increases in transient calcium levels lead to derepression of a negative feedback loop, allowing cell divisions to start. Preliminary results indicate a novel relationship between the speed of the initial calcium wave and the ability to achieve synchronous cell divisions.