Spatial epigenetic control of mono- and bistable gene expression

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Bistability in signaling networks is frequently employed to promote stochastic switch-like transitions between cellular differentiation states. Differentiation can also be triggered by antagonism of activators and repressors mediated by epigenetic processes that constitute regulatory circuits anchored to the chromosome. Their regulatory logic has remained unclear. A reaction-diffusion model reveals that the same reaction mechanism can support both graded monostable and switch-like bistable gene expression, depending on whether recruited repressor proteins generate a single silencing gradient or two interacting gradients that flank a gene. Our experiments confirm that chromosomal recruitment of activator and repressor proteins permits a plastic form of control; the stability of gene expression is determined by the spatial distribution of silencing nucleation sites along the chromosome. The unveiled regulatory principles will help to understand the mechanisms of variegated gene expression, to design synthetic genetic networks that combine transcriptional regulatory motifs with chromatin-based epigenetic effects, and to control cellular differentiation.
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