

$$S_t \circ c(h)(a_s)_{ti} c(s) + \mathfrak{S}_e m^i n(a_r)$$

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Stochastic gene expression and cell fate – or, why are identical twins so identical?

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Much recent work has shown that the process of gene expression is remarkably imprecise, leading to significant cell-to-cell variability in the numbers of mRNAs and proteins even in genetically identical populations. This raises a couple of questions: can cells exploit this variability for their own benefit? Conversely, do cells reduce the impact of variability in order to produce reliable outcomes in other contexts? We explored the first question by studying the random transitions of the soil bacterium *B. subtilis* to the "competent" state, tracing the origin of this transition to stochastic fluctuations in the expression of a regulatory gene. However, while unicellular organisms require stochastic variability to exist in multiple states simultaneously, one might expect multicellular organisms (with developmentally controlled cell types) to have mechanisms to tolerate this variability. To see if and how organisms do this, we are studying the gut formation pathway in embryonic development in *C. elegans*. Preliminary results show that the normal gut development pathway is remarkably robust, but this robustness can be destroyed by mutations that result in wildly varying embryonic fates. We are trying to link this variability to stochastic effects in the expression of developmental regulators caused by these mutations. We also present some results showing the randomness inherent to gene expression in mammalian cells.