Temperature-dependence of the single-cell variability in the kinetics of transcription activation in Escherichia coli

From in vivo single-cell, single-RNA measurements of the activation times and subsequent steady-state active transcription kinetics of a single-copy Lac-ara-1 promoter in Escherichia coli, we characterize the intake kinetics of the inducer (IPTG) from the media, following temperature shifts. For this, for temperature shifts of various degrees, we obtain the distributions of transcription activation times as well as the distributions of intervals between consecutive RNA productions following activation in individual cells. We then propose a novel methodology that makes use of deconvolution techniques to extract the mean and the variability of the distribution of intake times. We find that cells, following shifts to low temperatures have higher intake times, although, counter-intuitively, the cell-to-cell variability of these times is lower. We validate the results using a new methodology for direct estimation of mean intake times from measurements of activation times at various inducer concentrations. The results confirm that E. coli's inducer intake times from the environment are significantly higher, following a shift to a sub-optimal temperature. Finally, we provide evidence that this is likely due to the emergence of additional rate-limiting steps in the intake process at low temperatures, explaining the reduced cell-to-cell variability in intake times.

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Levosimendan alone and in combination with valsartan prevents stroke in Dahl salt-sensitive rats

The effects of levo-simendan on cerebrovascular lesions and mortality were investigated in models of primary and secondary stroke. We aimed to determine whether the effects of levo-simendan are comparable to and/or cumulative with those of valsartan, and to investigate whether levo-simendan-induced vasodilation has a role in its effects on stroke. In a primary stroke Dahl/Rapp rat model, mortality rates were 70% and 5% for vehicle and levo-simendan, respectively. Both stroke incidence (85% vs. 10%, P<0.001) and stroke-associated behavioral deficits (7-point neuroscore: 4.59 vs. 5.96, P<0.001) were worse for vehicle compared to levo-simendan. In a secondary stroke model in which levo-simendan treatment was started after cerebrovascular incidences were already detected, mean survival times were 15 days with vehicle, 20 days with levo-simendan (P=0.025, vs. vehicle), 22 days with valsartan (P=0.001, vs. vehicle), and 31 days with levo-simendan plus valsartan (P<0.001, vs. vehicle). The respective survivals were 0%, 16%, 20% and 59%, and the respective incidences of severe lesions were 50%, 67%, 50% and 11%. In this rat model, levo-simendan increased blood volume of the cerebral vessels, with significant effects in the microvessels of the cortex (ΔR=3.5±0.15 vs. 2.7±0.17 ml for vehicle; P=0.001) and hemisphere (ΔR=3.2±0.23 vs. 2.6±0.14 ml for vehicle; P=0.018). Overall, levo-simendan significantly reduced stroke-induced mortality and morbidity, both alone and with valsartan, with apparent cumulative effects, an activity in which the vasodilatory effects of levo-simendan have a role.

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