# A Mathematical Model of Cell State Dynamics in Melanoma

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Melanoma cells can transition between cell states, contributing to therapy resistance and immune evasion. These state changes involve dynamic and reversible shifts in gene expression, making it essential to understand the underlying regulatory mechanisms for developing effective therapies. We present a mathematical model of a minimal gene regulatory network comprising key transcription factors associated with melanoma cell states: MITF, ZEB1 and SOX10. Using deterministic temporal and spatio-temporal differential equation models, we analyse gene expression dynamics and classify stable states in a biologically meaningful way. We exploit an approximation, based on the number of transcription factors required for cooperative binding, which partitions regulatory dynamics into distinct regions in which the system behaves smoothly and steady states can be described analytically. At the population level, we use a naïve model of intercellular communication to explore how cells within a tumour can exhibit coordinated behaviour through travelling waves of gene expression. Additionally, we propose a method for deriving a condition that determines the final state of a population of communicating cells. This model provides a framework for better understanding some of the mechanisms driving gene expression dynamics during adaptation and melanoma tumour evolution and will ultimately inform and validate experimental hypotheses.