

D Improving Myeloma Thesis Phrasing



can you help me improve phrasing for this paragraph I am writing for my PhD thesis on myeloma dissemination? It's in latex code, please keep the syntax!

Third, one has to consider the role of the ECM in facilitating rapid adhesional plasticity. The ECM is known to regulate cell adhesion and migration rapidly, and its composition is altered by myeloma cells, including various mutated ECM proteins \cite{ibraheemBMMSCsderivedECMModifies2019, eversPrognosticValueExtracellular2023}. The ECM can also be remodeled by myeloma cells on site without requiring changes in cell surface factor expression, gaining one additional axis of potential adhesive interactions and improved flexibility for changing adherent sites.

Here's context:

Potential Mechanisms Facilitating the Fast Switch of Adhesion Dramatypes:
The hypothesis of rapid adhesional plasticity suggests that aggressive myeloma cells can swiftly alter their adhesion dramatype, as observed in $\text{INA-}\text{MSC}$ co-cultures. To facilitate such rapid changes, several molecular mechanisms might be utilized: Integrins can undergo rapid conformational changes from active to inactive forms, a process used to detach B-cell leukemia cells through small molecule treatment \cite{ruanVitroVivoEffects2022}. Additionally, myeloma cells can express proteases like heparanase to shed adhesion factors from their cell surface \cite{yangHeparanasePromotesSpontaneous2005}.

For INA cells, the author proposes three mechanisms that could explain this swiftness: First, $\text{NF-}\kappa\text{B}$ signaling is enriched in MAina cells. $\text{NF-}\kappa\text{B}$ is known as one of the fastest signaling pathways, capable of regulating the transcription of target genes within seconds \cite{gallego-sellesFastRegulationNFkB2022, zarnegarNoncanonicalNFkBActivation2008}. This signaling pathway is relevant for both *in vitro* experiments and MM patients \cite{sarinEvaluatingEfficacyMultiple2020}, making its downstream effectors robust targets for treatment.

Second, asymmetric cell division could explain the rapid loss of adhesion gene mRNA transcripts observed in MAina cells that emerged from MAina cells through cell division. This process was popularized by stem cell research for facilitating self-renewal \cite{shenghuiMechanismsStemCell2009}, yet its underlying molecular mechanisms are conserved in asymmetrically dividing cells and cellular polarization processes as well \cite{inabaAsymmetricStemCell2012, stjohnstonCellPolarityEggs2010}. Asymmetry can be established

\emph{intrinsically}, where factors are segregated between daughter cells, or \emph{extrinsically}, by placing two daughters into distinct microenvironments \cite{inabaAsymmetricStemCell2012}. It is debatable whether the definition of extrinsic asymmetric cell division is fulfilled by this work's observation of \nMAina daughter cells emerging out of range of \ac{MSC}, thereby delaying direct adhesion until the \nMAina re-attaches to an \ac{MSC}.

Intrinsic mechanisms could be explored by live-cell imaging of cell division events in \INA-\ac{MSC} co-cultures, followed by \textit{in situ} hybridization using fluorescently labeled antisense RNA probes. If successful, this could represent the first evidence for asymmetric cell division in MM, which could be useful for the popular \emph{cancer stem cell hypothesis}. However, \MAina cells do not currently fulfill the multipotency criterion required by stem cell terminology \cite{johansenMyelomaStemCell2016, liAsymmetricCellDivision2022}.



Certainly! Below is the revised version of your paragraph with improved phrasing, keeping the LaTeX syntax intact.

latex

Third, the role of the \ac{ECM} in facilitating rapid adhesional plasticity must be considered. The \ac{ECM} regulates cell adhesion and migration rapidly, and its composition is altered by myeloma cells, which include various mutated \ac{ECM} proteins \cite{ibraheemBMSCsderivedECMmodifies2019, eversPrognosticValueExtracellular2023}. Additionally, myeloma cells can remodel the \ac{ECM} on site without requiring changes in cell surface factor expression, providing an additional axis for potential adhesive interactions and improved flexibility in changing adherent sites.

Here is the context section with minor improvements for clarity and flow:

latex

\textbf{Potential Mechanisms Facilitating the Fast Switch of Adhesion Dramatypes:} The hypothesis of rapid adhesional plasticity suggests that aggressive myeloma cells can swiftly alter their adhesion dramatype, as observed in \INA-\ac{MSC} co-cultures. To facilitate such rapid changes, several molecular mechanisms might be utilized: Integrins can undergo rapid conformational changes from active to inactive forms, a process used to detach B-cell leukemia cells through small molecule treatment \cite{ruanVitroVivoEffects2022}. Additionally, myeloma cells can express proteases like heparanase to shed adhesion factors from their cell surface \cite{yangHeparanasePromotesSpontaneous2005}.

For \INA cells, three mechanisms are proposed to explain this swiftness: First, NF- κ B signaling is enriched in \MAina cells. NF- κ B is one of the fastest signaling pathways, capable of regulating the transcription of target genes within seconds \cite{gallego-sellesFastRegulationNFkB2022, zarnegarNoncanonicalNFkBActivation2008}. This signaling pathway is relevant for both \textit{in vitro} experiments and MM patients \cite{sarinEvaluatingEfficacyMultiple2020}, making its downstream effectors robust targets for treatment.

Second, asymmetric cell division could explain the rapid loss of adhesion gene mRNA transcripts observed in \nMAina cells that emerged from \MAina cells through cell division. This process, popularized by stem cell research for facilitating self-renewal \cite{shenghuiMechanismsStemCell2009}, has underlying molecular mechanisms conserved in asymmetrically dividing cells and cellular polarization processes as well \cite{inabaAsymmetricStemCell2012, stjohnstonCellPolarityEggs2010}. Asymmetry can be

established *intrinsically*, where factors are segregated between daughter cells, or *extrinsically*, by placing two daughters into distinct microenvironments [\cite{inabaAsymmetricStemCell2012}](#). It is debatable whether the definition of extrinsic asymmetric cell division is fulfilled by this work's observation of *maina* daughter cells emerging out of range of *acp{MSC}*, thereby delaying direct adhesion until the *maina* re-attaches to an *ac{MSC}*.

Intrinsic mechanisms could be explored by live-cell imaging of cell division events in *INA-ac{MSC}* co-cultures, followed by *in situ* hybridization using fluorescently labeled antisense RNA probes. If successful, this could represent the first evidence for asymmetric cell division in MM, which could be useful for the popular *cancer stem cell hypothesis*. However, *maina* cells do not currently fulfill the multipotency criterion required by stem cell terminology [\cite{johansenMyelomaStemCell2016, liAsymmetricCellDivision2022}](#).

These changes should help improve the clarity and readability of your thesis section.

ChatGPT kann Fehler machen. Überprüfe wichtige Informationen.