# Article information:

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<https://www.cell.com/cell-chemical-biology/fulltext/S2451-9456(19)30121-7?_returnURL=https%3A%2F%2Flinkinghub.elsevier.com%2Fretrieve%2Fpii%2FS2451945619301217%3Fshowall%3Dtrue>

# Article summary:

1. The activation of SRC kinase initially increases endothelial barrier function by organizing broad reticular adherens junctions and phosphorylating VE cadherin on Y731.

2. Prolonged SRC activity leads to disassembly of adherens junctions, resulting in increased endothelial permeability.

3. The RapR-kinase system is a useful tool for studying complex signaling systems and uncovering transient effects following protein activation.

# Article rating:

Appears moderately imbalanced: The article provides some useful information, but is missing several important points or pieces of evidence that would be required to present the discussed topics in a balanced and reliable way. You are encouraged to seek a more balanced perspective on the presented issues by exploring the provided research topics and looking at different information sources.

# Article analysis:

The article discusses the role of SRC kinase in regulating endothelial barrier function and highlights its time-variant effects. The study uses a protein engineering approach, the RapR-kinase system, to directly activate SRC in living cells and evaluate its effects on endothelial permeability. The results show that transient activation of SRC reduces endothelial permeability due to organization of broad reticular adherens junctions (AJs) and VE cadherin phosphorylation on Y731. However, prolonged SRC activation disassembles AJs, leading to an increase in endothelial permeability.

The article provides valuable insights into the complex behavior of SRC signaling in regulating endothelial barrier function. However, there are some potential biases and limitations that need to be considered. Firstly, the study only evaluates the effects of direct SRC activation using a synthetic biology tool and does not consider other factors that may influence endothelial permeability. Secondly, the study focuses on the role of VE cadherin phosphorylation in regulating AJs but does not explore other potential mechanisms involved in regulating endothelial barrier function.

Additionally, the article presents some unsupported claims regarding the role of SFK members in inducing distinct responses. While the study shows that activation of LYN kinase did not induce transient barrier enhancement like SRC, it is unclear whether this is a general characteristic of all SFK members or specific to LYN kinase.

Furthermore, there is some promotional content regarding the utility of synthetic biology tools in dissecting complex signaling systems. While these tools have undoubtedly contributed significantly to our understanding of cellular signaling pathways, their limitations and potential risks also need to be considered.

Overall, while the article provides valuable insights into the time-variant effects of SRC activation on endothelial permeability regulation, it is important to consider its potential biases and limitations when interpreting its findings.

# Topics for further research:

* Other mechanisms involved in regulating endothelial barrier function
* Role of other SFK members in inducing distinct responses
* Limitations and potential risks of synthetic biology tools
* Factors influencing endothelial permeability
* Signaling pathways involved in endothelial barrier function
* Role of other cadherins in regulating AJs

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