# Article information:

Signal-induced enhancer activation requires Ku70 to read topoisomerase1–DNA covalent complexes | Nature Structural & Molecular Biology
<https://www.nature.com/articles/s41594-022-00883-8>

# Article summary:

1. Transcriptional enhancers are a major molecular mechanism for cell-type and signal-specific transcriptional diversity.

2. Recent reports have revealed that active enhancers serve as hotspots for DNA single-strand breaks, or DNA nicks.

3. This article investigates the potential role of topoisomerase 1 (Top1) covalent complexes with genomic DNA (TOP1cc) in response to acute activating signals in signal-dependent enhancer activation.

# Article rating:

May be slightly imbalanced: The article presents the information in a generally reliable way, but there are minor points of consideration that could be explored further or claims that are not fully backed by appropriate evidence. Some perspectives may also be omitted, and you are encouraged to use the research topics section to explore the topic further.

# Article analysis:

This article is generally reliable and trustworthy, as it provides evidence from multiple sources to support its claims and conclusions. The authors provide a comprehensive overview of the current understanding of gene control by transcriptional enhancers, and discuss the potential role of TOP1cc in signal-dependent enhancer activation. The authors also cite relevant studies to back up their claims, such as those demonstrating that active enhancers serve as hotspots for DNA single-strand breaks, or DNA nicks, and those examining the role of Top1 at promoters. Furthermore, the authors use CUT&RUN assays to uncover the genomic landscape of TOP1cc in mammalian cells, which is an innovative approach that has not been used before.

The only potential bias in this article is that it does not explore any counterarguments or alternative explanations for its findings. While this is understandable given the scope of the article, it would be beneficial if the authors had discussed possible alternative explanations for their results or explored any counterarguments to their conclusions. Additionally, while this article does present both sides equally when discussing existing research on transcriptional enhancers and Top1 at promoters, it does not do so when discussing its own findings on TOP1cc in signal-dependent enhancer activation; instead, it focuses solely on supporting its own conclusions without exploring any other possibilities or counterarguments.

# Topics for further research:

* Transcriptional enhancer function
* DNA single-strand break hotspots
* TOP1 role at promoters
* Alternative explanations for gene control
* Counterarguments to TOP1cc findings
* CUT&RUN assays in mammalian cells

# Report location:

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