

# Length matters in gene expression

October 2 2012, by Lisbeth Heilesen

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A research team at Aarhus University reveals a surprising interplay between the ends of human genes: If a protein-coding gene is too short it becomes inactive! The findings also explain how some short genes have adapted to circumvent this handicap.

Human genomes harbour thousands of genes, each of which gives rise to proteins when it is active. But which inherent features of a gene determine its activity? Postdoctoral Scholar Pia Kjølhede Andersen and Senior Researcher Søren Lykke-Andersen from the Danish National Research Foundation's Centre for mRNP Biogenesis and Metabolism have now found that the distance between the gene start, termed the '[promoter](#)', and the gene end, the 'terminator', is crucial for the activity of a protein-coding gene. If the distance is too short, the gene is transcriptionally repressed and the output is therefore severely decreased. This finding outlines a completely new functional interplay between gene ends.

## Small genes utilise specialised terminators

Fortunately, most [human protein](#)-coding genes are long and are therefore not repressed by this mechanism. However, some genes, e.g. '[replication](#)-dependent histone genes', are very short. How do such genes express their information at all? Interestingly, many of these differ from the longer protein-coding genes by containing specialised terminators. And in fact, if such a specialised terminator replaces a normal terminator in a short gene context, the short gene is no longer transcriptionally repressed. It therefore appears that naturally occurring short genes have

evolved 'their own' terminators to achieve high [expression levels](#).

The new findings add to a complex molecular network of intragenic communication and help us to understand the basic function of genes.

The researchers behind the results that have just been published in the international journal *Genes & Development* are affiliated with the Danish National Research Foundation's Centre for mRNP Biogenesis and Metabolism at the Department of Molecular Biology and Genetics, Aarhus University.

**More information:** Promoter-proximal polyadenylation sites reduce transcription activity, [genesdev.cshlp.org/content/26/19/2169.full](https://genesdev.cshlp.org/content/26/19/2169.full)

Provided by Aarhus University

Citation: Length matters in gene expression (2012, October 2) retrieved 9 April 2024 from <https://medicalxpress.com/news/2012-10-length-gene.html>

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