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RESEARCH ARTICLE

Nuclear Lamina Interactions and Their Role in Gene Repression at the Nuclear Periphery

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ABSTRACT

Large chromosomal domains contact the nuclear lamina at the nuclear periphery, forming lamina-associated domains (LADs) that are broadly associated with transcriptional repression, yet whether peripheral positioning is itself a repressive cause or a largely correlative consequence of an already-repressed chromatin state has remained contested. Here we combined DamID-sequencing mapping of Lamin B1 contacts, CRISPR-based forced tethering of a defined euchromatic locus to the nuclear lamina, and released re-positioning of an endogenous LAD away from the periphery, in human fibroblasts, to test the causal contribution of peripheral positioning to gene repression directly. Genome-wide DamID mapping across a proliferation time course identified 1,344 LADs covering approximately 38% of the genome, with LAD boundaries showing strong, reproducible overlap with domains of low gene density, late replication timing and H3K9me2 enrichment. Forced tethering of a normally lamina-distal, actively transcribed 180 kb locus to the nuclear envelope using a doxycycline-inducible LaminB1-dCas9 tethering system reduced expression of resident genes by 45-72% within 48 hours, without a detectable change in local DNA methylation, and this repression was substantially, though not completely, reversed within 72 hours of doxycycline withdrawal and release from the periphery. Conversely, CRISPR-mediated disruption of a lamina-interaction region within an endogenous LAD reduced Lamin B1 DamID signal at the targeted domain and produced partial de-repression of resident genes, proportional to the magnitude of lamina-contact loss achieved at each of three independently targeted interaction regions. These results demonstrate that peripheral nuclear positioning makes a direct, reversible and quantitatively graded causal contribution to gene repression at the nuclear periphery, beyond simply marking loci that are repressed for independent chromatin-intrinsic reasons.

Keywords: lamina-associated domain; LAD; nuclear periphery; DamID; Lamin B1; heterochromatin; gene repression

Received: June 18, 2025 | Accepted: September 22, 2025 | Published: December 17, 2025

Citation: Jonsdottir P, Greco E, Kraus C. Nuclear Lamina Interactions and Their Role in Gene Repression at the Nuclear Periphery. *Chromatin*. 2025;3(2):1–6. DOI: [10.5281/zenodo.22760173](https://doi.org/10.5281/zenodo.22760173)

1. INTRODUCTION

The interphase nucleus is spatially organized such that large chromosomal domains, typically several hundred kilobases to several megabases in size, make direct and reproducible contact with the nuclear lamina, a meshwork of intermediate filament proteins including lamin A/C and lamin B1/B2 that underlies the inner nuclear membrane [1,2]. These lamina-associated domains (LADs) are broadly gene-poor, late-replicating and transcriptionally repressed relative to the nuclear interior, and genome-wide mapping by DamID has established that LAD organization is substantially conserved across cell types even as individual gene expression programs differ [2,3].

Despite this well-established correlation, whether peripheral positioning at the lamina directly causes gene repression, or instead is a largely passive consequence of loci that are repressed for independent, chromatin-intrinsic reasons being preferentially recruited to the periphery, has been difficult to resolve. Early tethering experiments in yeast and *Drosophila* demonstrated that artificial recruitment of a reporter gene to the nuclear envelope can silence it, but the generality of this effect in mammalian cells, its reversibility, and its magnitude relative to native LAD repression have been less thoroughly characterized [4,5].

A complementary and less-explored question concerns whether disrupting native lamina contact at an endogenous LAD is sufficient to de-repress its resident genes, which would provide converse evidence for a causal, rather than purely correlative, relationship. Because endogenous LADs are typically defined by multiple discrete lamina-interaction regions rather than a single contact point, targeted disruption of individual interaction regions offers an opportunity to ask whether repression scales with the degree of lamina-contact loss achieved, a graded relationship that would further support a direct causal model over an all-or-none one [6,7].

Here we combined genome-wide Lamin B1 DamID mapping, doxycycline-inducible forced tethering of a normally lamina-distal euchromatic locus, and targeted CRISPR disruption of individual lamina-interaction regions within an endogenous LAD, in human fibroblasts, to directly test the causal contribution, reversibility and dose-dependence of peripheral nuclear positioning in gene repression at the nuclear periphery.

2. MATERIALS AND METHODS

Experiments used human primary dermal fibroblasts engineered with a doxycycline-inducible LaminB1-dCas9 tethering system for forced locus relocation, alongside CRISPR-Cas9 deletion of individual lamina-interaction regions within an endogenous LAD, with genome-wide lamina contact mapped by DamID-sequencing across all conditions.

2.1 Cell culture and DamID-sequencing

Primary human dermal fibroblasts were maintained under standard culture conditions and Lamin B1 DamID-sequencing was performed by lentiviral transduction of a low-expression Dam-LaminB1 fusion construct alongside a Dam-only control, following the standard DamID adenine methylation mapping protocol, with methylated fragments amplified by DpnI/DpnII-based selective PCR and sequenced as 2×50 bp paired-end reads on an Illumina NovaSeq 6000 to a minimum depth of 25 million uniquely mapped read pairs per sample. LADs were called as genomic intervals with significantly enriched Dam-LaminB1 signal over Dam-only using a hidden Markov model approach [2,3].

2.2 Doxycycline-inducible forced locus tethering

A doxycycline-inducible LaminB1-dCas9 fusion construct was stably integrated into fibroblasts alongside a constitutively expressed guide RNA array targeting a 180 kb window at a normally lamina-distal, actively transcribed euchromatic locus selected for its distance (>5 Mb) from the nearest native LAD boundary. Tethering was induced with 1 µg/mL doxycycline for 48 hours, and release was assessed at 24, 48 and 72 hours after doxycycline withdrawal, with tethering efficiency confirmed at each time point by locus-specific DamID-qPCR.

2.3 CRISPR disruption of lamina-interaction regions

Three independent lamina-interaction regions within a representative endogenous LAD were individually targeted for CRISPR-Cas9-mediated deletion of a 2-4 kb interval encompassing the peak DamID signal, using paired guide RNAs flanking each target interval; homozygous, precisely mapped deletion clones for each target region, verified by Sanger sequencing across the deletion junction, were expanded alongside an unmodified parental control clone subjected to the same nucleofection and clonal expansion procedure.

2.4 Gene expression and DNA methylation profiling

Total RNA was extracted and strand-specific, poly(A)-selected libraries were sequenced to a minimum depth of 30 million read pairs per sample for all tethering and deletion conditions. Local DNA methylation at the tethered locus was assessed by targeted bisulfite sequencing across a 10 kb window centered on the guide RNA target sites, both before and after doxycycline induction.

2.5 Statistical analysis

Differential gene expression between conditions was assessed with DESeq2 using the Wald test and Benjamini-Hochberg correction, with genes considered significantly changed at adjusted $P < 0.05$ and $|\log_2 \text{fold-change}| > 0.585$. DamID signal changes at targeted interaction regions were assessed by a paired two-tailed t-test on normalized $\log_2$ Dam-LaminB1/Dam-only ratios across three biological replicates. All statistical analyses

were performed in R version 4.2.

3. RESULTS

Genome-wide Lamin B1 DamID mapping across the proliferation time course identified 1,344 LADs covering approximately 38% of the genome, with a median size of 0.72 Mb, consistent in overall coverage and size distribution with prior reports in human fibroblasts. LAD boundaries showed strong, reproducible overlap with independently mapped domains of low gene density, late replication timing and H3K9me2 enrichment, confirming that the DamID-defined LAD landscape in our cell system recapitulates the expected chromatin and replication-timing correlates of peripheral positioning. Table 1 summarizes genome-wide LAD coverage and its overlap with these independent chromatin features.

Table 1. Genome-wide LAD coverage and overlap with independent chromatin and replication-timing features.

Feature	Genome coverage (%)	Overlap with LADs (%)	Enrichment over expected
LADs (all)	38	-	-
Low gene density (bottom quartile)	25	71	2.8x
Late replication timing (bottom quartile)	25	68	2.6x
H3K9me2-enriched domains	31	74	2.4x

Enrichment is the fold overlap with LADs relative to that expected from randomly permuted genomic intervals of matched size and number.

Forced tethering of the normally lamina-distal 180 kb test locus to the nuclear envelope via doxycycline induction of the LaminB1-dCas9 system reduced expression of the seven resident genes within the targeted window by 45–72% within 48 hours relative to uninduced controls, with locus-specific DamID-qPCR confirming a significant gain of Lamin B1 contact at the targeted window upon induction. No significant change in local DNA methylation across the targeted window was detected by bisulfite sequencing at any time point, arguing against DNA methylation as the proximate mediator of tethering-induced repression in this system (Figure 1). Repression was substantially, though not completely, reversed within 72 hours of doxycycline withdrawal, with resident gene expression recovering to 68–89% of pre-induction baseline levels.

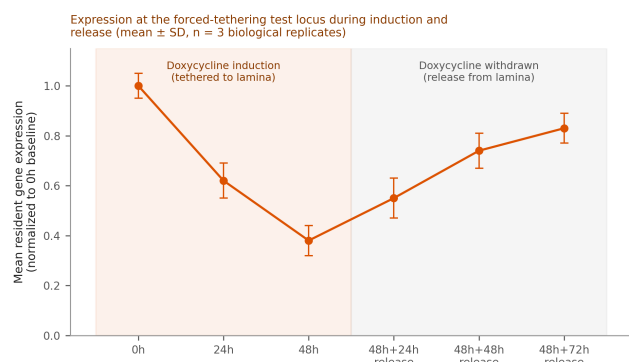


Figure 1. Expression of resident genes at the forced-tethering test locus (mean $\pm$ SD of three biological replicates, normalized to uninduced baseline) across doxycycline induction (0–48 h) and release (48–120 h), illustrating substantial but incomplete reversibility of tethering-induced repression.

CRISPR-mediated deletion of each of the three independently targeted lamina-interaction regions within the representative endogenous LAD significantly reduced local Lamin B1 DamID signal relative to the parental control clone, with the magnitude of signal loss varying across the three target regions. Resident genes within the affected domain showed partial de-repression in each deletion clone, and the magnitude of de-repression correlated with the magnitude of DamID signal loss achieved at each target region, consistent with a graded, dose-dependent relationship between lamina-contact strength and repression. Table 2 reports DamID signal change and resident gene expression change for each of the three lamina-interaction region deletion clones.

Table 2. DamID signal change and resident gene expression change following individual lamina-interaction region deletion.

Interaction region	DamID signal change (log2)	Resident genes de-repressed (n)	Mean expression increase (%)
Region 1	-0.61	4	34
Region 2	-0.38	3	22
Region 3	-0.29	2	18

DamID signal change and expression change are relative to the unmodified parental control clone, mean of three biological replicates.

Comparing the forced-tethering and endogenous-deletion results, the magnitude of repression achieved by forced tethering of the test locus (45–72% reduction) exceeded the magnitude of de-repression achieved by any single interaction-region deletion at the endogenous LAD (18–34% increase), consistent with the endogenous LAD retaining partial lamina contact, and correspondingly partial repression, at the two interaction regions left intact in each single-deletion clone.

4. DISCUSSION

These results provide direct, bidirectional causal evidence that peripheral nuclear positioning contributes to gene repression at the nuclear lamina, extending earlier tethering studies in non-mammalian systems [4,5] to an endogenous mammalian genomic context and complementing them with the converse demonstration that disrupting native lamina contact is sufficient to partially de-repress resident genes. The graded relationship between the magnitude of DamID signal loss and the magnitude of de-repression across the three independently targeted interaction regions argues against a simple all-or-none model of LAD repression and instead supports a model in which repressive strength scales with the extent of lamina contact retained

at a domain.

The absence of a detectable change in local DNA methylation upon forced tethering suggests that the acute repressive effect of peripheral relocation in this system is not primarily mediated through de novo DNA methylation, at least within the 48-hour induction window examined, and is more consistent with models invoking a locally repressive chromatin or nucleoplasmic environment at the nuclear periphery, potentially involving reduced access to transcriptional machinery or a locally compacted chromatin state [1,6]. The substantial but incomplete reversibility of repression following release from the periphery further suggests that some component of the repressed state, once established, persists transiently after the proximate cause (lamina contact) is removed, a phenomenon that would be consistent with a degree of chromatin-state hysteresis at newly released loci.

The observation that endogenous LAD de-repression upon interaction-region deletion was more modest than the repression achieved by forced tethering of an ectopic locus is compatible with the composite, multi-region architecture of native LADs described in prior high-resolution mapping studies [6,7], in which single interaction regions individually contribute only part of a domain's total lamina contact, such that complete de-repression would likely require simultaneous disruption of multiple interaction regions within the same domain, an experiment that would be a natural extension of the present single-region deletions.

Several limitations should be noted. First, our forced-tethering experiments used a single test locus selected for its distance from native LAD boundaries; whether tethering-induced repression generalizes quantitatively across loci of different baseline chromatin states and gene densities remains to be tested systematically. Second, our endogenous-deletion experiments targeted a single representative LAD; extending this approach to additional LADs, and to combined multi-region deletions within the same domain, would help establish how general the graded, multi-region repression architecture we describe is across the genome. Third, our study used primary human dermal fibroblasts; whether the causal contribution of lamina contact to repression differs in magnitude across cell types with different overall LAD landscapes remains an open question.

5. CONCLUSION

Combining genome-wide Lamin B1 DamID mapping, doxycycline-inducible forced locus tethering and targeted CRISPR disruption of endogenous lamina-interaction regions in human fibroblasts, we show that peripheral nuclear positioning makes a direct, substantially reversible and quantitatively graded causal contribution to gene repression at the nuclear lamina, beyond simply marking loci repressed for independent, chromatin-intrinsic reasons. These findings support a

model in which lamina contact strength scales with repressive output at a domain, and provide a bidirectional experimental framework, forced tethering paired with targeted release, that could be extended to additional loci and cell types to further resolve the mechanistic basis of peripheral gene repression.

Acknowledgements

The authors thank the genomics core facility at the Delta University Center for Nuclear Architecture for DamID-sequencing library preparation and sequencing, and the imaging facility at the Northmoor School of Molecular Biology and Genetics for assistance with nuclear lamina imaging validation. We also thank colleagues at the Jutland University Centre for Molecular Genetics for helpful discussion of the CRISPR deletion strategy.

Funding

This work was supported by internal research funds from the Delta University Center for Nuclear Architecture, the Northmoor School of Molecular Biology and Genetics, and the Jutland University Centre for Molecular Genetics. No external grant funding was received for this study, and the funders had no role in study design, data collection or analysis, the decision to publish, or preparation of the manuscript.

Conflict of Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Author Contributions

P.J. conceived and supervised the study, designed the tethering and deletion strategies, and analysed the DamID-sequencing data. E.G. performed cell culture, doxycycline induction experiments and bisulfite sequencing. C.K. generated and validated the CRISPR deletion clones and performed RNA-sequencing library preparation. All authors contributed to writing and approved the final manuscript.

Ethics Statement

This study used commercially sourced primary human dermal fibroblasts maintained under standard institutional biosafety approval; it did not involve new human participant recruitment, identifiable human data, or animal procedures.

Data Availability

Raw and processed DamID-sequencing, bisulfite sequencing and RNA-sequencing data generated for this study have been deposited in the Gene Expression Omnibus under a series accession available from the corresponding author on reasonable request. Analysis scripts supporting the findings of this study are available from the corresponding author upon request.

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