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# CRISPR/Cas9-Mediated Disruption of Targeted Genes in Gecko Oocytes Induces Biallelic Genomic Mutations

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#### Abstract

Researchers are studying the downstream effects of Fgf10 disruption on other genes and signaling pathways involved in limb development. By unraveling these intricate genetic cascades, scientists hope to gain a deeper understanding of the cellular processes and molecular interactions necessary for proper limb formation. The study of biallelic genomic mutations and Fgf10 knockout in gecko embryos offers valuable insights into the genetic basis of limb development. This research has implications not only for understanding the evolutionary significance of limb formation but also for regenerative medicine. Gecko species are known for their extraordinary regenerative capabilities, including the ability to regrow lost limbs. By elucidating the role of Fgf10 and other genes involved in limb development, scientists may unlock new strategies for promoting tissue regeneration and repair in humans.

Keywords: Genes • Gecko oocytes • Genomic mutations

### Introduction

The advent of CRISPR/Cas9 technology has revolutionized the field of genetic engineering, enabling precise and efficient genome editing in a wide range of organisms. In recent years, researchers have harnessed this powerful tool to study the functional role of specific genes in various animal models. This article focuses on the disruptive effects of CRISPR/Cas9-mediated genome editing on targeted genes in gecko occytes, unveiling the potential to unravel crucial genetic mechanisms underlying limb development. Specifically, we explore the biallelic genomic mutations introduced in F0 geckos and their correlation with limb defects, with a particular emphasis on the role of Fgf10 knockout.

#### **Literature Review**

CRISPR/Cas9 is a groundbreaking technology derived from the bacterial immune system that allows precise modification of DNA sequences. The system consists of two main components: the Cas9 endonuclease, which acts as molecular scissors, and a guide RNA (gRNA), which directs Cas9 to the specific target site within the genome. When Cas9 is guided to its target gene, it introduces Double-Strand Breaks (DSBs) at that location. The cellular repair machinery then attempts to fix these breaks, often leading to Small Insertions or Deletions (indels) that disrupt the gene's normal function [1].

Geckos, with their remarkable regenerative abilities and unique genetic traits, have emerged as valuable animal models for studying various biological processes, including limb development. By utilizing CRISPR/Cas9-mediated genome editing in gecko oocytes, researchers can explore the functional consequences of gene disruption during embryonic development. The targeted

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Received: 29 March, 2023, Manuscript No. Jgge-23-99990; Editor Assigned: 01 April, 2023, PreQC No. P-99990; Reviewed: 17 April, 2023, QC No. Q-99990; Revised: 22 April, 2023, Manuscript No. R-99990; Published: 29 April, 2023, DOI: 10.37421/2684-4567.2023.7.45

disruption of specific genes in gecko oocytes using CRISPR/Cas9 technology has yielded intriguing results. By introducing biallelic genomic mutations in F0 geckos, researchers have been able to investigate the phenotypic consequences of gene knockout. This approach has provided valuable insights into the importance of specific genes in gecko embryogenesis and has laid the groundwork for further investigations into the molecular mechanisms underlying limb development [2].

# Discussion

Among the genes targeted for disruption, Fgf10 has emerged as a key player in gecko limb development. Fgf10 encodes a signaling molecule that is critical for the growth and patterning of developing limbs. Through CRISPR/Cas9-mediated knockout of Fgf10, researchers have observed significant limb defects in gecko embryos, highlighting the gene's pivotal role in proper limb formation. These findings provide evidence for the functional importance of Fgf10 in gecko limb development and open avenues for further exploration into the genetic cascades involved in this intricate process.

The disruption of targeted genes in gecko oocytes using CRISPR/ Cas9-mediated genome editing has proven to be a valuable approach for studying gene function and unraveling the intricate mechanisms underlying limb development. The introduction of biallelic genomic mutations in F0 geckos, particularly through Fgf10 knockout, has shed light on the phenotypic consequences of gene disruption and enhanced our understanding of the genetic basis of limb defects. This research paves the way for future investigations aimed at deciphering the complex genetic pathways governing embryonic development and offers promising prospects for potential applications in regenerative medicine and evolutionary biology [3]. Genome editing technologies have provided scientists with unprecedented tools to explore gene function and uncover the intricate mechanisms underlying developmental processes. In recent research, the disruptive effects of biallelic genomic mutations introduced in F0 geckos, specifically through Fgf10 knockout, have shed light on the crucial role of this gene in limb development. This article delves into the fascinating findings of this study and highlights the implications for understanding limb formation in gecko embryos.

Genome editing, particularly the CRISPR/Cas9 system, has revolutionized the field of genetic engineering. It enables precise and targeted modifications of DNA sequences, allowing researchers to manipulate genes and study their functions in vivo. In the case of gecko embryos, CRISPR/Cas9mediated genome editing has proven to be a valuable tool for investigating the consequences of disrupting specific genes involved in limb development. By employing CRISPR/Cas9 technology, researchers have successfully introduced biallelic genomic mutations in F0 gecko embryos. Biallelic mutations refer to alterations that occur in both copies of a targeted gene. This disruption triggers changes in the gene's sequence and function, ultimately leading to observable effects on the developing gecko embryos [4].

Fgf10, a gene encoding a fibroblast growth factor, plays a critical role in limb development across various species. It acts as a signaling molecule, orchestrating the growth and patterning of developing limbs. Through targeted Fgf10 knockout using CRISPR/Cas9, researchers have uncovered a link between the absence of Fgf10 and limb defects in gecko embryos. These defects manifest as malformations, alterations in limb size, and disruptions in the patterning of limb structures.

The identification of limb defects resulting from Fgf10 knockout in gecko embryos has prompted further investigation into the underlying molecular mechanisms. Researchers are studying the downstream effects of Fgf10 disruption on other genes and signaling pathways involved in limb development. By unraveling these intricate genetic cascades, scientists hope to gain a deeper understanding of the cellular processes and molecular interactions necessary for proper limb formation [5]. The study of biallelic genomic mutations and Fgf10 knockout in gecko embryos offers valuable insights into the genetic basis of limb development. This research has implications not only for understanding the evolutionary significance of limb formation but also for regenerative medicine. Gecko species are known for their extraordinary regenerative capabilities, including the ability to regrow lost limbs. By elucidating the role of Fgf10 and other genes involved in limb development, scientists may unlock new strategies for promoting tissue regeneration and repair in humans [6].

### Conclusion

Biallelic genomic mutations, particularly through Fgf10 knockout, in F0 gecko embryos has revealed significant limb defects, providing compelling evidence for the critical role of Fgf10 in limb development. This study exemplifies the power of genome editing technologies in unraveling the

complexities of gene function and developmental processes. The findings not only deepen our understanding of gecko limb formation but also hold promise for advancing regenerative medicine research. Further exploration of the molecular mechanisms underlying limb defects may pave the way for innovative therapeutic approaches and inspire future studies on limb regeneration in diverse organisms.

# Acknowledgement

None.

## **Conflict of Interest**

None.

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How to cite this article: Luis, Fernando. "CRISPR/Cas9-Mediated Disruption of Targeted Genes in Gecko Oocytes Induces Biallelic Genomic Mutations." *J Genet Genom* 7(2023): 45.