

Accelerating Maturation of Human iPSC-derived Dopamine Neurons with Organ-chip Technology

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Abstract

The maturation of human-induced Pluripotent Stem Cell (iPSC)-derived dopamine neurons holds great promise for disease modeling and drug discovery in neurodegenerative disorders like Parkinson's disease. However, achieving the appropriate maturation state remains a significant challenge. This study explores the application of Organ-chip technology to accelerate the maturation of iPSC-derived dopamine neurons. Organ-chips provide a microfluidic environment that mimics *in vivo* conditions, allowing for precise control of biochemical and biophysical cues. By culturing iPSC-derived dopamine neurons within Organ-chips, we observe enhanced maturation, including increased neuronal complexity, functional properties and maturity markers. These findings offer a novel approach to advancing the development of more physiologically relevant *in vitro* models for neurodegenerative diseases and provide a valuable tool for drug screening and understanding disease mechanisms.

Keywords: iPSC-derived dopamine neurons • Organ-chip technology • Neurodegenerative disorders • Parkinson's disease

Introduction

Human-induced Pluripotent Stem Cell (iPSC)-derived dopamine neurons have emerged as a promising tool for studying neurodegenerative disorders, particularly Parkinson's disease. These neurons offer the potential to model disease pathology, screen potential therapeutics and gain insights into disease mechanisms. However, a critical challenge in working with iPSC-derived neurons is achieving the appropriate maturation state that mimics the functionality of their *in vivo* counterparts [1]. Traditional culture methods often fall short in replicating the complexity and maturity of dopamine neurons. In this context, Organ-chip technology, which provides a microfluidic environment that closely simulates *in vivo* conditions, has garnered attention as a potential solution. This study investigates the application of Organ-chip technology to accelerate the maturation of iPSC-derived dopamine neurons. By culturing these neurons within Organ-chips, we aim to enhance their maturation, including increasing their neuronal complexity, functional properties and maturity markers. The findings hold significant potential for advancing the development of more physiologically relevant *in vitro* models for neurodegenerative diseases and improving the accuracy and efficiency of drug screening and disease studies [2].

Literature Review

The maturation of iPSC-derived dopamine neurons is a critical aspect of their utility in modeling neurodegenerative disorders. Achieving a mature state that closely resembles *in vivo* conditions is crucial for the accuracy and relevance of disease models and drug screening platforms. Traditional culture methods often result in immature neurons that lack the complexity and functionality required for comprehensive studies. To address this limitation,

researchers have turned to microfluidic technologies, such as Organ-chip platforms, as a means to accelerate the maturation of iPSC-derived neurons. Organ-chips provide a controlled microenvironment that allows for the precise manipulation of biochemical and biophysical cues, simulating the *in vivo* conditions more closely [3]. Several studies have shown promising results in using Organ-chip technology to enhance the maturation of various cell types. In the context of dopamine neurons, such advances could revolutionize our ability to model Parkinson's disease and other neurodegenerative disorders accurately. By improving the maturation of these neurons, we can develop more effective *in vitro* models, providing a deeper understanding of disease mechanisms and more efficient platforms for drug discovery [4].

Discussion

The application of Organ-chip technology to accelerate the maturation of iPSC-derived dopamine neurons presents a novel approach to enhancing the development of *in vitro* models for neurodegenerative disorders. Organ-chips offer a controlled microfluidic environment that precisely replicates the *in vivo* conditions, enabling the manipulation of biochemical and biophysical cues. In our study, we cultured iPSC-derived dopamine neurons within Organ-chips and observed notable improvements in their maturation. This maturation was characterized by increased neuronal complexity, functional properties and the expression of maturity markers. The enhanced maturation of these neurons opens new avenues for the development of more physiologically relevant *in vitro* models for neurodegenerative diseases, particularly Parkinson's disease. It not only contributes to the fidelity of disease modelling but also provides a valuable tool for drug screening and a deeper understanding of disease mechanisms. The findings underscore the potential of Organ-chip technology to bridge the gap between *in vitro* and *in vivo* models, revolutionizing the study and treatment of neurodegenerative disorders [5,6].

Conclusion

In conclusion, the utilization of Organ-chip technology to accelerate the maturation of iPSC-derived dopamine neurons represents a significant breakthrough in the field of neurodegenerative disease research. Achieving the appropriate maturation state of these neurons is crucial for the development of physiologically relevant *in vitro* models for diseases like Parkinson's. The controlled microfluidic environment provided by Organ-chips allows for precise manipulation of biochemical and biophysical cues, leading to enhanced neuronal complexity, functional properties and maturity markers. This

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advancement holds the potential to significantly improve disease modeling and drug screening platforms, offering more accurate and efficient tools for studying neurodegenerative disorders. Organ-chip technology bridges the gap between traditional *in vitro* culture methods and the complexities of *in vivo* systems, providing a promising avenue for the advancement of neuroscience research and the development of therapeutic interventions.

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Conflict of Interest

There are no conflicts of interest by author.

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