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Basic Science

Abstract Title

Investigating the role of Fan1 and Mecp2 in Neurodevelopmental Health and Disease

Background

Neurodevelopment is a complex process, and disruptions can result in severe neurological conditions. One of these conditions is Rett Syndrome (RTT). RTT is a neurodevelopmental condition caused by mutations to the MECP2 gene. RTT primarily affects girls and leads to the loss of previously acquired skills. One key aspect of neurodevelopment is the ability to make connections and repair damage. Two genes Mecp2 and Fan1 are important for building and repairing the brain.

Objective

This research aims to explore if changes to Fan1 can help rescue function after a loss of Mecp2. Overall, this research aims to provide a better understanding of how different parts of the brain work together during development, how Fan1 and Mecp2 interact and what happens when these interactions are disrupted.

Methods

Mosaic Analysis with Double Markers (MADM) will be used. MADM is a powerful tool for studying neurodevelopment as it utilizes fluorescence and recombination to create genetically mosaic mice that can be analyzed at single-cell resolution. Cells are identified as either mutant (green) or wildtype (red) and can then be compared across and between mouse models to analyze changes in morphology, location and number of cells in the different layers of the brain.

Results

Current progress has shown that at both postnatal day 0 (P0) and P21 interneuron output is reduced when Fan1 is mutated compared to controls. Mouse models are being generated to investigate the impact of this mutation in excitatory neurons. Investigations into mouse models of RTT are ongoing.

Conclusion

The results indicate that a loss of Fan1 leads to a decreased number of cortical interneurons. It is unknown if this is due to a defect in proliferation, migration or production. Further investigations are being conducted to uncover when these changes begin to occur and to gain a better understanding of the mechanisms involved.

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