

This study examines the molecular and cellular differences between individuals with Alzheimer's disease and healthy subjects, focusing on gene activity, cellular function, and RNA expression levels.

### **Gene Activity Imbalance in Alzheimer's Disease**

Data from Log Fold Change (LFC) analysis (Q4\_Figure\_1 and Q4\_Figure\_3) shows that Alzheimer's neurons exhibit significant differences in gene activity. Genes like *RASGEF1B* (LFC 2.16) and *LINGO1* (LFC 1.807) are upregulated, while genes such as *SNHG14* (LFC -2.499) and *MEG3* (LFC -1.802) are downregulated. Q4\_Figure\_2 reveals that approximately 13,000 genes display increased activity, whereas 8,000 show decreased activity in Alzheimer's cells, suggesting a disruptive gene activity imbalance that may destabilize neural processes.

### **Impact on Cellular Function**

Effect size analysis (Q4\_Figure\_1) highlights further disruptions. Genes with large positive effect sizes, such as *SLC26A3* (4.089) and *RASGEF1B* (3.637), indicate enhanced cellular functions, while genes with negative effect sizes, like *SNHG14* (-1.509), suggest suppressed cellular activities. These opposing trends suggest that Alzheimer's disease alters normal cellular function, over-stimulating some areas while hindering others, which could affect brain function and resilience.

### **Reduced RNA Expression in Alzheimer's Neurons**

The RNA count comparison (Q2\_Figure\_1) further underscores cellular decline in Alzheimer's patients. The median RNA count is lower, and data distribution is narrower compared to healthy individuals, indicating decreased RNA expression. This reduction could impair Alzheimer's neurons' ability to maintain necessary cellular processes, potentially impacting cognitive function.

### **Conclusion**

In summary, Alzheimer's disease introduces a profound imbalance in gene activity, disrupts cellular functions, and reduces RNA expression, leading to impaired neural communication. These findings, supported by visual data (Q4\_Figure\_1, Q4\_Figure\_2, Q4\_Figure\_3, and Q2\_Figure\_1), illustrate the cellular-level challenges posed by Alzheimer's and highlight critical molecular alterations that may inform therapeutic approaches.