# HNRNPU-related disorder

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Generating effective therapies for neurodevelopmental disorders has remained elusive. An emerging drug discovery approach for neurodevelopmental disorders is to characterize transcriptome-wide dysregulation in an appropriate model system and screen therapeutics based on their capacity to restore functionally relevant expression patterns. We characterized transcriptomic dysregulation in a human model of

HNRNPU

-related disorder to explore the potential of such a paradigm. We identified widespread dysregulation in functionally relevant pathways and then compared dysregulation in a human model to transcriptomic differences in embryonic and perinatal mice to determine whether dysregulation in an

in vitro

human model is partially replicated in an

in vivo

model of

HNRNPU

-related disorder. Strikingly, we find enrichment of co-dysregulation between 45-day-old human organoids and embryonic, but not perinatal, mice from distinct models of

HNRNPU

-related disorder. Thus, hnRNPU deficient human organoids may only be suitable to model transcriptional dysregulation in certain cell types within a specific developmental time window.