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Environment and neuronal activity dependent midbrain dopamine neurotransmitter plasticity: Potential new therapeutic approaches for midbrain dopamine imbalances

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Too much or too little midbrain dopamine neurotransmission causes symptoms associated with several prominent brain and behavioral disorders (e.g. Parkinson's disease, schizophrenia, depression, ADHD, OCD and drug addiction). We have recently helped discover a new form of adult brain plasticity which presents as different numbers of dopamine neurons in mice and humans exposed relatively briefly (days-weeks) to different environments. Specifically, we see 10-20% more or fewer midbrain dopamine neurons in mice paired with the opposite sex for 1 week, or housed in enriched environments (EE; i.e. toys, running wheels, etc.) for 2 weeks. These changes are dependent on neuronal activity because they can be induced solely by administration of ion-channel agonists or antagonists directly into midbrain for 2 weeks, and EE-induction of more midbrain dopamine neurons is abolished by blocking synaptic transmission in midbrain. Moreover, we have evidence that the same occurs in adult humans. Specifically, there are 4-fold more midbrain dopamine neurons in postmortem brains of people who died in summer compared with winter, and this occurs without any changes in the total number of midbrain neurons or markers of cell death. Most recently, we have obtained evidence indicating this environment-induced 'midbrain dopamine neurotransmitter plasticity' affects midbrain dopamine-dependent behavior in adult mice. Together, our data suggest that exposing patients exhibiting symptoms of midbrain dopamine imbalances to appropriate environmental stimuli might help alleviate those symptoms. Furthermore, uncovering cellular and molecular mechanisms of midbrain dopamine neurotransmitter plasticity might lead to new drug-based therapeutic approaches.

Biography

Tim D Aumann has completed his BSc (Honors) and PhD from Monash University, Australia. He completed Post-doctoral Training at Monash University from 1995 to 1998 and the University of Washington, USA from 1999 to 2003 where he undertook studies into the neural control of movement. From here, he returned to the Florey Institute of Neuroscience and Mental Health at The University of Melbourne, Australia, to study midbrain dopamine neurons in rodent models of Parkinson's disease. He currently heads research into midbrain dopamine neurotransmitter plasticity at the Florey. He has published 30 papers in reputed journals and serves as an Editorial Board Member of PLOS ONE.

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