Nurr1: A Novel Target for the Treatment of Catecholamine Related Diseases

INVENTION:
Investigators at the Salk Institute have found that the nuclear receptor Nurr1 induces expression of the enzyme tyrosine hydroxylase in both undifferentiated and differentiated mammalian cells, including adult hippocampal progenitor cells. This results in increased tyrosine hydroxylase activity, which in turn increases the production of the catecholamine neurotransmitter molecules norepinephrine and epinephrine. There are certain disease states caused by deficiencies in these neurotransmitters, such as Parkinson's disease, manic depressive disorder, and schizophrenia. Therefore, these findings reveal Nurr1 as a potential target for therapeutics to treat those disorders.

APPLICATIONS:
• Experimental platform to model tyrosine hydroxylase-related human disease
• Identification of tyrosine hydroxylase-related deficiencies
• Potential treatment for catecholamine-related deficiencies, such as Parkinson's disease, manic depressive disorder and schizophrenia

ADVANTAGES:
The discovery provides a new target for development of novel treatments for diseases caused by deficiencies in catecholamines.

STAGE OF DEVELOPMENT: Preclinical in vitro data

BACKGROUND:
During development, neural stem cells differentiate into the different types of neurons and glia found in the adult central nervous system (CNS) and peripheral nervous system (PNS). These different types of neurons are classified based on the particular types of neurotransmitters they produce. For example, dopaminergic neurons produce dopamine, while noradrenergic neurons produce norepinephrine. The neurotransmitters dopamine and norepinephrine belong to a class of compounds called catecholamines, which are derived for the common metabolite tyrosine. The rate-limiting step for the production of these catecholamines is the conversion of tyrosine to DOPA by the enzyme tyrosine hydroxylase. The findings of Salk's investigators reveal the regulation of this process by the nuclear receptor Nurr1.

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PATENT STATUS: U.S. Patent 6,312,949


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TECHNOLOGY ID: RD9459