Neurons, in the adult mammalian Central nervous system (CNS), in contrast to other somatic cells of the body are highly differentiated cells that have lost their ability to proliferate. Despite the fact that this essential difference helps to maintain synaptic contacts and thereby important neural networks, perturbations beyond tolerable limits provoke irreversible functional deficits.

Damage to the neuronal circuitry by way of neuronal loss occurs as a result of various factors. Genetic factors, drugs and the process of ageing predispose the subtle network of neurons to degenerate. All neurodegenerative disorders are characterized by gradually evolving, slow, relentless neuronal death unaccompanied by an intense tissue reaction. There is selective loss of certain defined groups of neurons that might be related either anatomically or physiologically. The onset of these diseases is often subtle and progression is insidious. These diseases are exemplified by Parkinson’s disease, Alzheimer’s disease, Huntington’s chorea and Amyotrophic lateral sclerosis.
A substantial body of evidence suggests the involvement of environmental toxins in the etiology of various neurodegenerative disorders. Although, not conclusively, Aluminium and Manganese have been implicated in the onset of Alzheimer's and Parkinson's disease respectively.

Lead, one of the ancient known neurotoxin, has been shown to cause neurologic injury, to reduce nerve conduction velocity and to lead to mental retardation and hyperactivity in humans as well as animals especially during the developing period. Experimental studies indicate iron deficiency as an important factor influencing the severity of lead toxicity.

The vulnerability of the developing nervous system during the perinatal period is attributed to rapid ontogenesis of different cell types, their division, differentiation, migration, proliferation and aggregation into brain regions, development of synaptic connectivity and myelination. Transplacental movement and poor development of the blood-brain-barrier coupled with absence and/or incomplete maturation of specific organ system(s) and xenobiotic metabolising enzymes further make the developing stages more prone to the toxic action of lead.

Lead, also remains a public health problem because strategies for decreasing the body burden of lead such as chelation therapy, are currently controversial. Evidence indicating that some chelating agents can mobilise lead from bone and redistribute it to soft tissue target organs including brain has been reported. Moreover, at present an insufficient basis exists to support the contention that chelation treatments actually reverse lead-induced behavioural manifestations.

The cell replacement strategy of neural transplantation was developed in an attempt to overcome the intrinsic limitation of the neurons to divide and replace lost cells. Through successful cell replacement the damaged brain can be reconstructed to a certain extent, in order to compensate for neuronal loss and the neurological deficits thereof.
The use of fetal tissue transplantation, in recent years has been successfully carried out in humans as well as experimental animals to reverse neurological deficits.

In the present investigation the toxicological implications of perinatal exposure to lead under conditions of iron deficiency have been studied. This was followed by fetal neural transplantation as an experimental tool to further determine the precise underlying mechanism(s) of neurochemical and behavioural deficits by the extent of reversibility of the damage.