Maya Karginova

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# The Paradox of Neural Stem Cell Transplantation and Anthropogenic Air Pollution

**Abstract**

Both neuroscientists and victims of neurodegenerative diseases share enthusiasm for emerging neural stem cell (NSC) transplantation as a therapy for Alzheimer's and Parkinson's disease. Simultaneously, researchers causationally link air pollutants to neurodegeneration. As incidents of these diseases have increased in the past few decades and are projected to increase in the decades to come, we must question the consensual integrity of NSC transplantation and observe the paradox of regenerative therapy within the climate of human-caused air pollution.

## Neural Stem Cell Transplantation as a Therapy for Neurodegenerative Disease

NSC transplantation, a modern regenerative therapy, introduces cells that replace damaged neurons and glial cells and integrate into the established, complex electrochemical circuit of the human brain. Via secretion of neurotrophic factors, NSCs modulate inflammation, therefore granting opportunity to form synapses and directly replace tissue (McGinley et al. 18). NSC transplantation may act as a preventative for further neurodegeneration by mitigating neuroinflammation (De Gioia et al. 20). The following successes of stem cell transplantation offer hope to patients with Alzheimer's and Parkinson's disease.

Transplanting fetal cortex-derived neural stem cells into murine models of Alzheimer's, McGinley et al. found improvement of short-term memory and reduction of amyloid plaque in mice (McGinley et al. 18). Since then, cognitive improvements have also been observed by transplants of induced pluripotent stem cells (iPSCs). Research by Sawamoto et al. found increased dopamine production and decreased motor symptoms of Parkinson's in human patients after transplanting dopaminergic

progenitors derived from iPSCs (Sawamoto et al. 25). These findings represent NSCs' powerful potential in regenerative medicine.

**Responsibility and Risk of Stem Cell Transplantation**

Such radical processes of stem cell harvesting, cultivation, and implementation call for scrutiny. Given transplanted cells must appropriate the microenvironments inside the mind, and these microenvironments may vary from mind to mind, the patterns and predictability of differentiation raise concerns. Is differentiation and function of transplanted cells guaranteed? iPSCs can form teratomas, and tumorigenic risks of iPSCs define the observed margin of error of NSC transplantation (Liu et al. 13). This margin may likewise exceed neuroethical boundaries of changing personality and identity as a result of NSC transplantation.

With the capacity to form new synapses, NSC transplantation alters patients' minds physically and emotionally. Such change is irreversible. When administering therapy to cognitively compromised patients, such as those with Alzheimer's and Parkinson's, how can responsibility and risk be consensual? Consent by proxy may be the anticipated solution, though, the moral weight of consenting to a stem cell transplant is greater when the stem cells may alter the identity of the patient (Akabayashi et al. 19).

**Air Pollutants and Neurodegenerative Disease**

The need for regenerative therapies may be attributed to the rise of neurodegenerative disease. Nearly 7 million Americans currently live with Alzheimer's disease, a figure projected to double by 2050 (Alzheimer's Association 2025). Parkinson's disease affected 11.77 million people globally in 2021 and is forecast to exceed 25 million by 2050, a 112% increase (Dickinson 25). A nonconsensual neurodegenerator must be further investigated: anthropogenic air pollution.

Alongside a progressive industry for neurodegenerative remedy is a climate of neurodegenerative exacerbation. Air pollution increases risk of both Parkinson's and Alzheimer's disease, not merely through respiratory harm, but through direct neurobiological mechanisms. Fine particulate matter, PM2.5, accelerates the same amyloid and tau pathology that defines Alzheimer's: each 1 $\mu g/m^3$ increase in ambient PM2.5 is associated with a 19% higher risk of worsening amyloid and tau aggregation (Kim et al. 25). PM2.5 likewise elevates risk of Parkinson's disease dementia 17% per unit increase (Zhang et al. 25).

Explaining how inhaled agents impair cognitive development requires mapping the biological routes by which particles move from the air to the brain. Around 50% of inhaled ultrafine particles are deposited in the lungs, pass through the capillary barrier, and enter the surrounding tissue (Meo 22). Pollutants can move into the bloodstream, spreading to organs like the heart and brain. Another route taken is directly through the olfactory nerve pathways into the brain (Fu et al. 22). This translocation of particles induces neuroinflammation and oxidative stress that damage neurons, impair synapse formation, and alter microglial activity in regions central to cognition, such as the prefrontal cortex, hippocampus, and olfactory structures (Meo 22). Prolonged exposure to environmental pollutants increases risk for neurodegenerative disease. Longitudinal epidemiology and neuropathological research increasingly link chronic air-pollution exposure with elevated incidence of Alzheimer's disease, Parkinson's disease, and cerebrovascular complications (Calderón-Garcidueñas et al. 25). Notably, even modest increases in ambient oxidants, such as ozone, have been associated with higher relative risks of Alzheimer's. A 211% risk of increase of contracting Alzheimer's per every 10 ppb of O3 was found, stressing the unavoidable consequences of air chemistry (Jung et al. 21).

**The Nonconsensual Neurodegenerator**

The aforementioned limitations of consent to NSC transplantation from an individual with a neurodegenerative disease is recalled by a similar nonconsensual cause of neurodegenerative disease: anthropogenic air pollution.

Consent presupposes alternatives. A choice requires options. The polluted air of industrial corridors, disproportionately concentrated in low-income communities, enters lungs that have no mechanism to refuse it. A child growing up adjacent to a refinery did not consent to elevated amyloid production. A grandmother, developing Alzheimer's, did not consent to the particulate matter worsening her tau pathology. These exposures are structural. Consent, as a moral framework, assumes a subject with the means to say no. Environmental neurotoxicity removes that option before the subject understands what is being taken.

**The Paradox of Regenerative Therapy and Anthropogenic Air Pollution**

If stem cell transplants can reduce neuroinflammation and amyloid aggregates, the rise of Alzheimer's and Parkinson's rates can, theoretically, be slowed. Must we correct the same neurodegeneration we environmentally induce?

Society produces the neurotoxin and the treatment. It funds pharmaceutical research and industrial emissions. It asks cognitively compromised patients, who may not be able to meaningfully consent, to receive irreversible neural transplants as remedy for a condition that was, in part, imposed without their consent.

**Conclusion**

To develop increasingly sophisticated neural interventions while expanding the industrial conditions that necessitate them is not progress. Society is stepping onto the treadmill of neurodegeneration. The ethical significance of this paradox extends beyond medicine. It reveals a

disconnect between technological innovation and environmental responsibility. Neural stem cell transplantation may be ambitiously effective against neurodegeneration, but it does not resolve the underlying question of justice raised by preventable neurotoxic exposures. As regenerative therapies continue to advance, neuroethical debate must expand beyond the clinic to include the environmental conditions that shape public health.

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