Methamphetamine Use as a Developmental Factor in Parkinson's Disease - Yes or No?

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Introduction

Parkinson's disease (PD) is a neurodegenerative disease manifested clinically through bradykinesia, tremor and / or resting stiffness. These symptoms are caused by the irreversible loss of dopamine (DA), a monoaminergic neurotransmitter whose loss occurs from the degeneration of the dopaminergic neurons located in the substantia nigra (SN).[1,3]

The term Parkinsonism refers only to the motor manifestations described above, however, it does not mention the underlying cause of the symptoms. Thus, it becomes a broader term than the PD itself. This is reflected by the prevalence of the disease, where parkinsonism is more prevelent in relation to PD. [1]

Methamphetamine (MA) is a psychostimulant drug whose effects are expressed in the central and peripheral nervous system. MA is related to a moderate increase in the risk of developing PD and Parkinsonism. This is due to its neurotoxicity that affects monoaminergic neurotransmission, increasing the time of exposure of DA in the synaptic cleft, since it can not be recaptured. [1-3]

The aim of this study is to understand if there is any association between methamphetamine use and the development of Parkinson's disease.

Methodology

For the accomplishment of this work we analyzed scientific review articles through the online platform PubMed.
Search terms: “Methamphetamine”; “Neurodegenerative Disease”; “Parkinson Disease”.
Of the articles found we have selected only the most recent (2015-2019) and the most relevant ones for the topic under discussion.

Parkinson's disease:

- Rare in subjects <50 years;
- The prevalence in individuals with ≥ 65 years is in the order of 2-3%;
- ± 10% of PD cases have a known genetic cause;
- The risk increases with age and with the successive loss of dopaminergic neuronal integrity, which is initially exponential.[1]

The dopaminergic integrity is not possible to measure in living individuals but the hyperecogeneity of NS can be used as a potential marker. This pathology of NS is found in 80 to 90% of individuals with PD. It is also a prediction of the future development of PD since it has been shown that individuals over 50 years of age are 17 times more predisposed to develop PD in 3 years than individuals without hyperechogenicity of SN. In individuals who consume MA, a greater area of hyperechogenicity of SN was observed.[1]

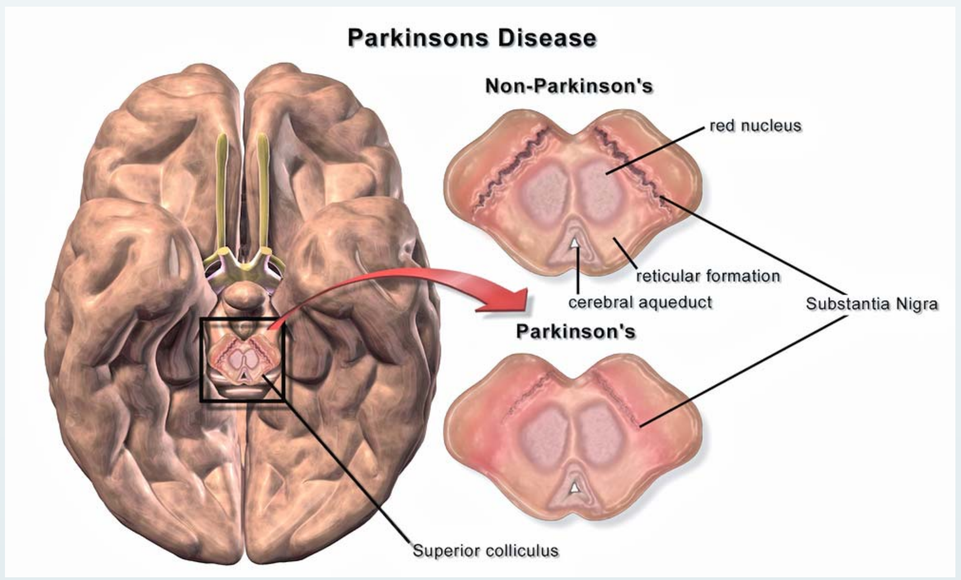

Figure 1: Comparation between the SN of individuals without PD and the abnormal morphology of SN in individuals with PD.

https://lighthouserecoveryinstitute.com/does-meth-cause-parkinsons/

The brain structures most damaged by the chronic consumption of MA are the striatum and SN, which are part of the nigrostriate dopaminergic system. The imbalance of this system is the reason for cognitive and motor damages present in PD,such as the lack of fine motor, which are present in PD.[1-3]

MA consumption has neurotoxic effects:

- Causes an increase in the release of monoamines, such as DA, from dopaminergic neurons in the brain;
- It inhibits DA uptake in the synaptic cleft by blocking the DA transporter (DAT), increasing its exposure time;
- It inhibits the storage of DA in the synaptic vesicles and increases its concentration at the cytosolic level.[1-3]

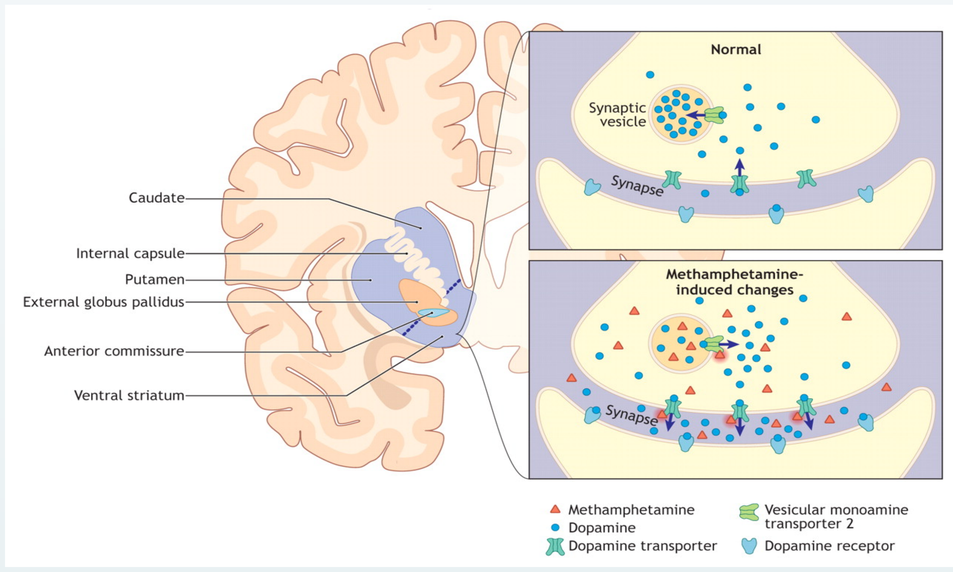

Figure 2: Effects induced by MA consumption on dopaminergic neurons.

Stephen J. Kish. Pharmacologic mechanisms of crystal meth.2008 Jun 17.

Chronic MA consumers have DA levels in the striatum reduced by up to 50%, the caudate nucleus reduced by 61% and the putamen by 50%. Further evidences show that these consumers suffer a decline in the amount of SN, like in individuals with PD, which may indicates that MA consumers are susceptible to develop neurodegerative diseases such as PD.[1,2]

Factors that can mask the effects of MA:

- The age at onset and abstinence from MA use: young individuals have better results after a neurological injury than older individuals because they have greater neuroplasticity and better cognitive reserve.
- Individuals with smoking habits may have the neurotoxic effects of MA attenuated by nicotine, reducing or delaying the onset of parkinsonism.[1]

Conclusion

The progressive loss of dopaminergic function caused by the comsumption of MA may reach the threshold of loss of dopamine necessary for the clinical manifestation of PD. It is then verified that MA plays an important role in the development of PD.

References

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