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# The Neurological Impact of the Green Tea (Camellia sinensis) Catechin EGCG on Parkinson's Disease

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## ABSTRACT

Parkinson's disease (PD) is a slow-onset neurodegenerative disease, the second most prevalent, after Alzheimer's disease. In all neurodegenerative diseases, neuronal degeneration is inevitable and results in the depletion of function in several regions of the brain, causing the symptoms of such diseases. Though it is prevalent, Parkinson's disease currently has no cure. Due to this lack of a cure, for decades, researchers have been either trying to find a cure for the life-changing disease, or they have been trying to discover approaches to mitigate the symptoms and side effects of PD. One such approach that has been looked into, is the consumption of green tea by patients battling PD. Green tea, scientifically known as Camellia sinensis, contains several catechins, a type of polyphenol (naturally found compounds in many beverages), which demonstrate several beneficial properties, many being neuroprotective. The most prominent catechin found in green tea is epigallocatechin-3-gallate (EGCG), known for its health benefits. This article aims to explore and analyze studies done regarding general green tea components and EGCG, including its neuroprotective implications on Parkinson's disease. Both in vitro and in vivo studies have been done to examine the overall benefits of green tea on PD. Such benefits include a reduction in symptoms of PD, reduced risk of PD development, and overall protection against neuronal damage: factors that this study aims to explore.

## Introduction

Parkinson's disease, also known as PD, is a neurodegenerative disease that occurs from the loss of dopamine-producing neurons in the *substantia nigra pars compacta*, a region in the basal ganglia of the brain. The basal ganglia are a group of nuclei in the brain, responsible for motor coordination, meaning that damage to this area leads to mainly motor impairments (Meade et al., 2019). PD itself however also involves cognitive impairments (Cleveland Clinic, 2020). Some examples of both motor and cognitive impairments associated with PD include tremors, slowness of movement, memory impairment, and more. As the disease progresses, dementia-like symptoms and depression are also likely to arise (Cleveland Clinic, 2020). PD poses a significant health issue globally, impacting millions worldwide. Though there is no cure for Parkinson's, many treatments are available to assist with alleviating the symptoms of the disease (Cleveland Clinic, 2020). With its prevalence, a copious amount of research is being done to find affordable and natural remedies to mitigate the impact of the disease's symptoms. One such possible natural remedy is a popular ancient beverage: green tea. Research suggests that green tea, rich in antioxidants and polyphenols, may be beneficial in managing the symptoms of diseases. The potential effects of green tea on Parkinson's opens up a whole new area of research regarding natural remedies to the disease.

Several research studies have shown the association between PD and green tea consumption. In an article published by Neurology Asia in Qingdao, China, the claim that was concluded was that tea consumption played a big role in reducing the risk of developing PD. The article mentioned a study in Hong Kong, China, that further explored the association between general tea drinking and PD (Zhen et al., 2019). The results found

that those who drank less than one cup of tea per day had an OR of 1.51, indicating that their odds of having PD were 1.51 times more than those who drank tea more regularly, having one or more cups of tea per day (Chan et al., 1998). Though not referencing the type of tea or why the tea reduces the risk of obtaining PD, these results support the basic idea that drinking tea has positive impressions regarding a reduced risk of development of PD.

Concerning green tea specifically, and in order to answer why the beverage can reduce the risk of PD, several studies have been conducted and analyzed in research articles. One such article published by *The Science of Parkinson's* develops the idea that the consumption of EGCG has the great ability to prevent alpha-synuclein from assembling into fibrils (Simon, 2016). Alpha-synuclein is a protein that holds a large concentration in each brain cell. Alpha-synuclein aggregation is directly correlated to the brain of those with Parkinson's. Once these proteins aggregate, Lewy bodies form, serving as an indicator for PD. Specific to PD, the presence of a-synuclein is especially harmful in the mitochondria, where it can cause oxidative stress and decrease the production of ATP. This loss of energy production can interrupt the vesicles that store dopamine, leading to dopamine oxidative stress. Through in vitro experimentation, it was found that green tea polyphenol epigallocatechin-3-gallate (EGCG) was able to inhibit the production and aggregation of a-synuclein. However, ECGC can not only prevent the formation of alpha-synuclein fibrils (aggregated form) but it can also bind to the alpha-synuclein protein and allow it to function as a safe and non-toxic aggregate (Simon, 2018). These findings support the hypothesis that EGCG and PD can be related and that EGCG can assist in the long-term treatment of PD.

According to one study, using a PD mice model, EGCG was administered to the mice orally and was found to have profound effects. These effects included the neuronal cell death rate in the mice falling under 50% (Pervin et al., 2018 ). By reducing neuronal cell death through oral implications of EGCG, this study reflects that oral consumption of green tea can have significant impacts on neurons. Specific to PD, by reducing neuronal death, dopaminergic neurons can also be spared, allowing patients with PD to experience a reduction in harmful symptoms in regards to motor impairments and tremors, caused by the loss of dopamine.

With more than ten million people worldwide living with PD, and the number just increasing by the day, extensive research and experimentation must be done in order to find a cure for the neurodegenerative disease. At the least, researchers should look for more affordable and accessible forms of treatment to mitigate the effects of PD (Marras et al., 2018). Given that holistic and natural forms of treatment are being looked into, it is worth examining the correlation between green tea specifically and PD, in order to verify the impact that consuming tea can have on PD patients. The findings correlating green tea to PD can be life-altering for those who do not have the means to purchase any other form of expensive remedy to relieve their symptoms or reduce their risk. This unique approach to treatment not only places emphasis on using diet to intervene in neurodegenerative disease management, but it also provides a natural means of treatment to those who prefer it.

## Methodology

The primary goal of this research was to analyze and determine the benefits that green tea consumption can have on patients with Parkinson's disease. The classification of the type of research conducted in this article is a secondary literature review which is formed based on informational experimentation and research-based articles. The analysis method utilized in this research paper was qualitative in order to determine the benefits of green tea and its components on patients with PD. Through the process of gathering data, several studies and experiments were analyzed regarding PD in general and how compounds such as EGCG impact the cells of those with the disease, both in vitro and in vivo. Physical tools and experimentation were not used to conduct this research, as all information has been obtained from online sources. Bias was also reduced and avoided by making use of articles from various countries and various research journals each with different purposes.

## Green Tea and EGCG

Green tea, scientifically known as Camellia sinensis, is a beverage that has been consumed for thousands of years worldwide, first emerging in China. China and Japan are just two of the many nations that promote green tea consumption for medicinal purposes. The popular beverage is largely known for its catechin content which is greater than that of wine, apples, red grapes, and chocolate. Through significant research, green tea has been found to assist in helping cardiovascular complications, cancer risk, and stress (Prasanth et al., 2019). Similarly, green tea has also been proven to help alleviate the effects of neurodegenerative diseases and their associated risks, like PD.

One such cause of the relief caused by green tea comes from one of its many components, epigallocatechin-3-gallate (EGCG). EGCG is a catechin, a type of compound found in plants. Catechins are a subgroup of a larger plant compound group called polyphenols, found in abundance in green tea. EGCG is associated with many potential health benefits: cognition, mood, blood pressure, cancer prevention, cholesterol, and more (Brownstein, 2024). One of the most notable health benefits that EGCG is proven to have is its impact on neurodegenerative diseases.

The main purpose of this literary review is to analyze the impact of the EGCG compound, from green tea, on Parkinson's disease. More specifically, this paper will focus on EGCG and its effects on dopamine, motor function, and alpha-synuclein (a very important and significant protein involved in PD).

## Alpha-Synuclein and MPTP

In order to measure and do experiments on the impacts of various factors on PD, researchers first had to determine how they were going to model the disease and what they would be measuring and focusing on through their experimentation to verify or reject their hypotheses.

Alpha-synuclein, known for its linkage to PD, has become one of the key research factors in any PD-related experimentation. Alpha-synuclein is a protein that makes up an abundant amount of the proteins in a nerve cell. Alpha-synuclein in healthy neuronal cells is expressed as unfolded branch-like structures extending out from the neuron, allowing them to transfer neurotransmitters between neurons. In their natural state, alpha-synuclein is unfolded, they can then however aggregate into oligomers that are stabilized by beta-sheet structures, and then aggregated further into fibrils. Thus far, five known mutations of alpha-synuclein are linked to PD. More specifically, what has been found in patients with PD, is that alpha-synuclein misfolding has occurred, which is what causes the protein to clump up in aggregates and form Lewy bodies. Lewy bodies are a clear indicator of PD, and are found in regions of the brain that suffer neuronal loss, many of which have neurons that produce dopamine. Alpha-synuclein is thought to be passed between neurons, causing Lewy body formation in other brain regions, supporting the disease's progression (*Parkinson's and Alpha-Synuclein*, n.d.). To summarize, alpha-synuclein aggregation eventually leads to cell death in neurological pathways, like with dopamine neurons.

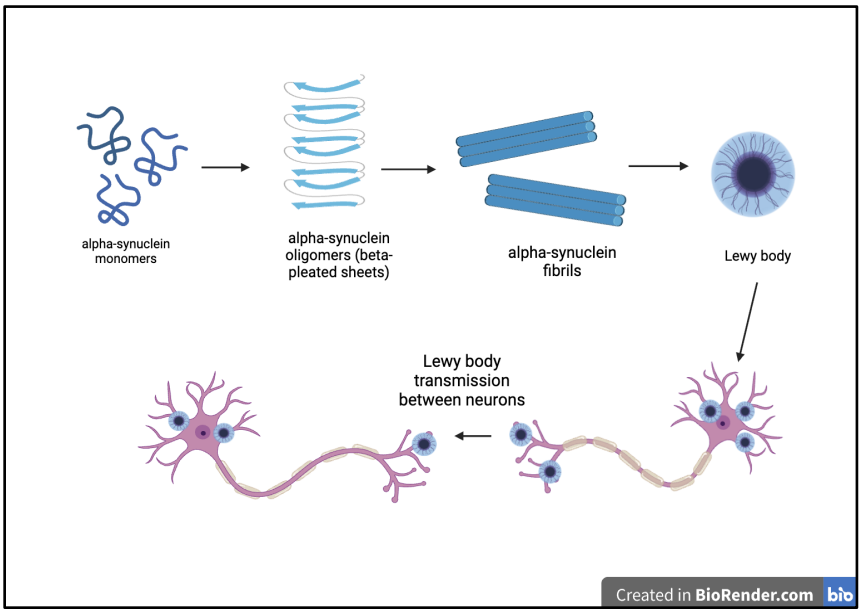


**Figure 1.** Alpha-synuclein aggregation in PD. Source: Meghna Kapa (Created with BioRender.com). Description: Alpha-synuclein aggregation is a key factor in the progression and development of Parkinson's disease In the neurons in the brain, the alpha-synuclein monomers first misfold into beta-pleated sheet-like structures to form the alpha-synuclein oligomers. These oligomers then further aggregate into fibrils which make up the Lewy bodies. The Lewy bodies are then transmitted between neurons which assists in the progression and spread of the neurodegenerative disease.

Now that researchers and scientists knew of alpha-synuclein's significant role in PD, they had to determine how to reflect the disease in both in vivo and in vitro models, so that proper experimentation could be performed for research purposes. A specific toxin, 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine, known as MPTP, served as one of many factors of reflection for PD. MPTP was found to target neurons specific to PD. MPTP itself is not toxic, but once it is oxidized to form MPP+, this new form is in fact toxic. MPTP is able to easily enter brain cells, where it is then captured by organelles like lysosomes of astrocytes (a subtype of glial cells). These astrocytes contain monoamine oxidase (MAO-B) which then aids in the conversion of MPTP to MPP+. Once MPP+ comes into contact with extracellular fluid, it is transported to dopamine nerve terminals where it can cause dopaminergic neuronal loss(Sian et al).

## In Vitro Studies

The cause of Parkinson's is not entirely known, which makes it more difficult to find a cure and treatment, however, several in vitro studies (studies done in a test tube) have been conducted concerning the relationship between the green tea catechin EGCG and other related aspects of PD specifically. Catechins are a form of flavonoid. One of the most prominent catechins in green tea is epigallocatechin-3-gallate, or EGCG. This catechin has been studied heavily to conclude its neuroprotective properties. Green tea, in particular, contains almost four times the concentration of catechins than black tea (Mandel et al., 2008). Due to this finding, it is supported that green tea is the most appropriate and most effective form of tea in mitigating PD symptoms.

In one specific in vitro study, it was found that on P12 cells, EGCG was able to prevent the aggregation of alpha-synuclein. As previously mentioned, this aggregation of alpha-synuclein is caused by misfolding and mutations that are related to PD. In those with PD, some of the alpha-synuclein is in the mitochondria where it

suppresses Complex-1 activity. Complex-1 inhibition causes oxidative stress, which dopamine neurons are subject to, causing them to stop producing dopamine, the main cause of PD. EGCG is able to bind to the alpha-synuclein and rearrange the alpha-synuclein structure to prevent the creation of toxic fibrils, thus preventing PD risk and progression (Malar et al., 2020).

In another study in vitro, the effect of EGCG on already-made aggregate forms of alpha-synuclein was analyzed. The experiment included adding equal concentrations of EGCG to the alpha-synuclein fibrils. The results found were that EGCG was able to rearrange the fibrils after incubation, resulting in shorter and less harmful protein aggregates (Bieschke et al., 2010). This rearrangement of the fibrils means that alpha-synuclein aggregation can be mitigated, and further, the spread of these toxic compounds can be transformed into benign compounds, with dopamine inhibition also being prevented.

More specifically, the way that EGCG is able to bind to the alpha-synuclein protein and prevent its toxicity is through hydrophobic and hydrogen-bonding interactions with the EGCG compound and the alpha-synuclein protofibrils which are beta-pleated. Through studies, it has been determined that EGCG disrupts the beta-sheet structure in alpha-synuclein protofibrils (Yao et al., 2020). This binding to the beta-sheet structure consequently creates smaller protein aggregated which leads to a loss in function to aggregate and become toxic (Korgiopoulo, 2020).

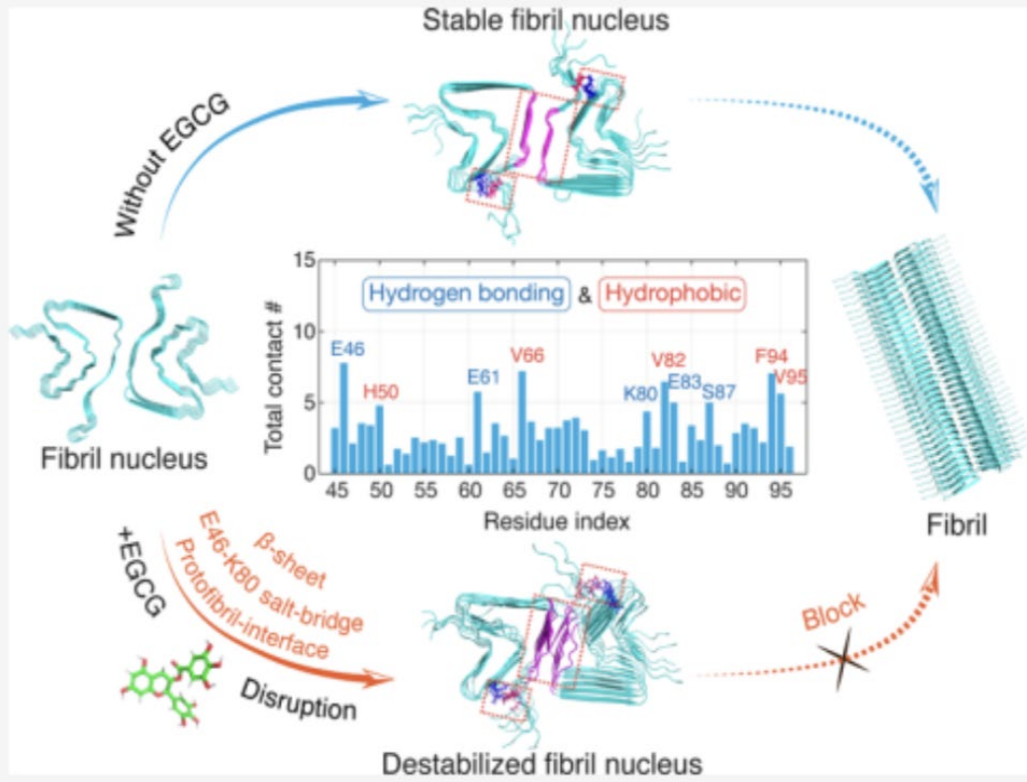


**Figure 2.** Impact of EGCG on fibril structure and production through disruption of beta-pleated sheets. Source: Yao et al., 2020. Description: The protofibril structure without the addition of the green tea catechin EGCG produces a further stable protofibril which will then go on to produce a functional and complete fibril. The protofibril with the addition of EGCG caused a disruption to the beta-pleated sheets that are present in the alpha-synuclein protofibril structure. This disruption causes a more destabilized protofibril structure which is then unable to produce a functional alpha-synuclein fibril that can aggregate and form Lewy bodies.

## In Vivo Studies

In addition to studies done in vitro, some studies have also been done on animals like mice and rats in relation to the green tea and PD correlation. In one specific study, EGCG was given to mice for ten days. Additionally, the compound MPTP was also administered with EGCG for another four days. MPTP, as previously mentioned, is a neurotoxin that causes dopamine neurodegeneration, an indicator of those with PD. The results showed that the EGCG administered with the MPTP was found to increase the amount of dopamine that was supposed to be depleted by the MPTP (Levites et al., 2001). This finding supported the idea that in vivo, EGCG is able to bolster dopamine concentration, combatting and overriding the impact of dopamine depletion that is regular in patients with PD.

An additional enzyme, monoamine oxidase-B (MAO-B), is also heavily correlated with PD, specifically by converting MPTP to its oxidized and toxic form, MPP+. An increase in this enzyme has been proven to be linked to patients with PD, yet, the inhibition of this enzyme leads to prevention of the inhibition of dopamine. As previously stated, PD is caused by a lack of dopamine transfer between neurons and the synaptic cleft. Thus, MAO-B inhibition supports treatment for PD. EGCG not only is able to bind to alpha-synuclein to prevent aggregation but it is also found to inhibit the presence of MAO-B in aged rat brains, further accentuating its purpose against PD (Malar et al).

In another experiment done with mice, the benefits of EGCG on PD symptoms were further supported. The experiment involved the previously mentioned MPTP-induced mice once again. These mice were then administered with EGCG for twenty days. The impact of the EGCG was measured through a test called the "pole test". This test was assessed every five days of the EGCG treatment and ultimately aimed to measure the motor coordination of the mice. The set-up of the experiment involved a pole that was taped fifty centimeters high, with a cork ball at the top, the mice were then placed looking directly upwards at the ball and the time for the mouse to look downward was measured as a reflection of their motor ability. The results found were that the time for the mouse to look downward was significantly increased after being given MPTP, however, the time was reduced once EGCG was administered ( Zhou et al., 2018). This finding is meant to support that EGCG consumption is able to reduce the bradykinesia symptom of PD, a symptom that causes PD patients to move with difficulty or with increased slowness.

Supportingly, a 2015 study analyzed the effects of EGCG on the 6-OHDA model of PD in rats. The neurotoxin 6-hydroxydopamine, known as 6-OHDA, is used as a reflective model of PD to cause dopaminergic neuronal loss in rat models of PD. This neurotoxin is administered into the rats through injection and the dopamine loss it causes further is able to reflect PD-like motor impairments (*6 OHDA Parkinson Model, Animal Model of Parkinson's | Melior*, n.d.). In this study, the 6-OHDA induced rats went through multiple tests, to measure the impact of various compounds on their motor ability, one such compound being EGCG. One of the tests conducted was an open-field test, aimed at measuring the rats' locomotor ability since motor impairments are prominent in those with PD. The test placed the rats in a small wooden box divided into four quadrants, which was later illuminated with a red light. The number of times the rats crossed with all four paws into each quadrant was then measured to reflect their locomotor activity. The results of the study found that the control group (the 6-OHDA rat models alone) had a statistically significantly fewer number of crossings into other quadrants (over five-minute intervals) than the 6-OHDA group that was given EGCG (Natália Bitu Pinto et al., 2015). These results support that in the 6-OHDA model of rats, EGCG is also seen to have positive effects on motor function related to the motor impairment symptoms of the disease.

## Discussion

Through the in vivo and in vitro evaluations of EGCG and its ultimate impact on PD, it is clear that there is a positive correlation between the two factors, supporting that consumption of green tea has positive effects on PD. Through analysis of the in vitro studies, it has been found that EGCG is able to bind to the beta sheets in alpha-synuclein and alter its form to prevent further misfolding. This prevention of misfolding is directly linked to the reduction of alpha-synuclein aggregation and toxicity, a prominent cause of PD. Additionally, green tea includes four times the amount of catechins than black tea, which further signifies the importance of green tea specifically for patients with PD. Additionally, through in vivo studies, the impacts of EGCG on live PD models have been shown to prove significant benefits. EGCG for one has been revealed to reduce the amount of MPTP and MAO-B in PD rat models. These reductions further correlate with reductions in dopaminergic neuronal loss, a main contributing factor of PD. Looking at motor impacts, EGCG has also been shown to increase motor movements through the 6-OHDA model in rats and the MPTP-induced rat models. These results, combined, are increasingly significant in highlighting the effects of EGCG on several factors of PD, both on the protein level and on the level of motor symptoms.

## Conclusion

Parkinson's disease, or PD for short, is a disease that is caused by a decrease in dopamine-related neurons in the substantia nigra, a structure part of the basal ganglia, which is in charge of movement and motor cognition. This depletion of neurons is what leads to a lack of motor function and can later lead to memory loss and signs of depression. With no known cure found for Parkinson's, there has been an increase in support for more research regarding natural remedies and methods of alleviation for neurodegenerative diseases. Through copious amounts of research, the abundant green tea catechin epigallocatechin-3-gallate (EGCG) has gained significant attention due to its neuroprotective properties and linkage to PD, AD, and other neurodegenerative diseases.

The studies that have been referenced in this text have consistently supported that the EGCG catechin has been able to substantially decrease the damage caused by dopaminergic neurons and the aggregation of alpha-synuclein. By binding to the beta-pleated sheets of alpha-synuclein and restructuring the protein, EGCG was able to ultimately reduce the toxicity of the substance's aggregation in the brain, mitigating the effects of PD and its risk factor. EGCG also has some impact on other diseases like Alzheimer's, due to its ability to decrease the presence of tau and amyloid fibril formation. The linkage between EGCG and PD is significant enough with respect to alpha-synuclein for further research to be done. Something that was not present in the studies analyzed that could have been present is an analysis of the effects of EGCG in humans or primates like apes, to support the effects to a more real scale. Another factor that was not included in the analyzed texts was the potentially harmful effects of EGCG and whether or not the compound could be counterproductive in its use. Since there is abundant research supporting its benefits, future research should be done to analyze the impact that EGCG has in clinical trials, specifically on humans. EGCG can also be researched further to determine its more medicinal use, outside of green tea consumption (potentially in capsules), to be correlated with more specific dosages to enhance its function.

## Limitations

The research in this paper aims to analyze the effects of green tea on Parkinson's disease. Since this literary review is secondary, primary articles and experimentation results were able to be used to analyze the relationship between the two factors of research. The paper seeks to delve into the in vivo and the in vitro studies done specifically regarding EGCG and PD models. The research also more specifically details the role of EGCG in

the symptoms and risk factors of PD. As a result of this research exploring a clear correlation between EGCG and PD, mainly in terms of reducing alpha-synuclein aggregation, there can be further research done to explore more medicinal-related benefits of EGCG, rather than simply through green tea. The scope of this paper is that it only focuses on the neurological impact of the green tea catechin EGCG on aspects of PD. This research does not however look into the potential harmful aspects of EGCG or the potential counteractivity of the substance. A challenge faced while conducting this research was that there were very limited sources and information on this specific topic due to the amount of research done regarding green tea and PD.

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