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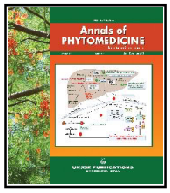

Review Article : Open Access

An overview of the various medicinal plants used in the treatment of Parkinson's disease

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Abstract

Over 10 million individuals worldwide are afflicted with Parkinson's disease (PD), the most common degenerative neurological movement sickness. The declining dopaminergic (DA-ergic) function with time in the brain's substantia nigra pars compacta (SNpc) region is the defining feature of PD; the etiology of the illness is yet unknown. The main culprits are thought to be oxidative damage and mitochondrial malfunction. The PD treatment options accessible today mostly uses Levodopa that, which has a lot of side effects but may be able to partially slow down the progression of the disease. It is yet unknown which possible medication could be used to cure or stop the illness. Since both are necessary for proper brain functioning, it is clear that redox stability and mitochondrial function replenishment appear to be key therapeutic approaches against PD. Vast amount of studies in this area have demonstrated the neuroprotective and anti-apoptotic potential of certain natural and synthetic products through the enhancement of mitochondrial activity and reduction of oxidative stress. Consequently, the goal of this review is to talk about some of the major natural medicinal plants: *Curcuma longa* (L.), *Gingko Biloba* (L.), *Withania somnifera* (L.) Dunal, *Bacopa monnieri* (L.) Pennell, *Mucuna pruriens* (L.) DC and *Camellia sinensis* (L.) Kuntze in relation due to their potential for neuroprotection. In addition, new treatment approaches for PD are being developed.

1. Introduction

The most common degenerative neurological movement illness, Parkinson's disease (PD) affects more than 10 million people worldwide. The condition is more common as people age. With an estimated 4% of cases identified before the age of 50, men are more prone than women to have PD (Wooten *et al*., 2004). Furthermore, the average annual cost of medicine for PD patients is approximately $2500, and the potential cost of therapeutic surgery for PD patients is approximately $100,000. In addition, the global population of PD sufferers is growing tremendously, which has a negative impact on a person's social and personal life (Van Den Eeden *et al*., 2003). PD is defined neuropathologically by Lewy bodies pathology, which is caused by gradual degeneration of DA-ergic neurons, especially in the brain's SNpc region (Spillantini *et al*., 1997). The degraDAtion of DA-ergic neurons is thought to be the funDAmental cause of the characteristic classical motor and non-motor symptoms (Khoo *et al.,* 2013). Also, the possibility of getting PD is multifactorial and heterogeneous, with an unclear etiology possessing changes in genetic components, environment, age, gender, and ethnicity as risk factors. Research has shown that altered mitochondrial malfunction and oxiDAtive stress are the two important indicators of cellular stress that are connected to PD etiology (Simon-Sanchez *et al.,* 2007). Studies have shown that for the brain to function at its best, mitochondrial quality control and dynamics are essential; any alterations cause neuronal cell death, which has been compiled into a comprehensive overview. This is seen in animal models and the brain postmortem samples from PD patients, where it was discovered that complex activity, or mitochondrial function, was reduced (Bose and Beal, 2016). Additionally, mitochondria participate in mitophagy, which checks for malfunctioning organelles. Additionally, by distributing organelles optimally in response to the maintenance at the terminal end of neurons, synaptic action is enhanced by mitochondria, which meet the energy requirements of neurons (Hauser and Hastings, 2013). Consequently, one of the main targets that may result in the death of neuronal cells in neurodegenerative diseases is mitochondria. Though research has made significant progress in the previous several years, our current understanding of the disease's origin is still very limited. Consequently, one of the main targets that may result in the death of neuronal cells in neurodegenerative diseases is mitochondria (Jiang *et al*., 2016). Although, research has made significant progress in the previous several years, our current understanding of the disease's origin is still very limited. Additionally, it is given along with carbidopa, a peripheral decarboxylase inhibitor. This aids in reducing the negative effects of L-DOPA, which primarily involves cardiovas-cular and gastrointestinal issues (Michel *et al*., 2016). The application of monoamine oxidase-B (MAO-B) inhibitors is an additional PD treatment (Ono *et al*., 2001). Because DA metabolism increases oxidative stress and mitochondrial dysfunctions, the MAO-B enzyme is more active. Neuronal cell death is thought to be caused by altered oxidative stress and mitochondrial malfunction

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as of yet (Connolly, 2014). Thus, in the current context, treatment strategies that maximize reactive oxygen species (ROS) and improve mitochondrial function are being carefully evaluated. An organic extract derived from therapeutic herbs is the pharmaceutical strategy being studied in this context, and it has been demonstrated to have a positive impact on PD (Jadiya *et al*., 2011). Even still, the precise molecular mechanism of action is still unknown. Nonetheless, it is thought that these natural plant compounds' primary goal should be to maximize the formation of ROS (Khuwaja *et al*., 2011). Over the past few decades, research has identified a large number of plants that have therapeutic potential against neurodegenerative disorders such as PD, Alzheimer's disease, and others (Nagashayana *et al*., 2000). Thus, this review aims to discuss some of the medicinal plants: *Curcuma longa* (L.), *Gingko Biloba* (L.), *Withania somnifera* (L.) Dunal, *Bacopa monnieri* (L.) Pennell, *Mucuna pruriens* (L.) and *Camellia sinensis* (L.) Kuntze in relation to their potential for neuroprotection and the creation of new therapeutic approaches against Parkinson's disease (Van der Merwe *et al*., 2017).

Table 1: Above table shows the various medicinal plant and chemical constituent present in them

S.No.	Herbs	Common name	Chemical constituent
1	*Mucuna pruriens* (L.)	Velvet bean	Levodopa, glycoside, gallic acid, levodopa, and glutathione
2	*Curcuma longa* (L.)	Turmeric	Curcumin
3	*Camellia sinensis* (L.) Kuntze	Green tea	Catechins and polyphenols
4	*Gingko Biloba* (L.)	Maidenhair tree	Ginkgolide B
5	*Bacopa monnieri* (L.)	Brahmi	Bacoside and Bacopaside
6	*Withania somnifera* (L.) Dunal	Ashwagandha	Withaferin and withanolide
7	*Camellia sinensis* (L.) Kuntze	Black tea	Theaflavins

2. Herbs used for Parkinson's disease

2.1 Velvet bean: *Mucuna pruriens* (L.)

A tropical bean plant with medicinal use is called *M. pruriens* (Mp). Mp seeds are used to treat nephropathy, ulcers, and helminthiasis because of their anti-inflammatory properties. The use of Mp in PD Dates back thousands of years, as it was once employed in Ayurveda medicine to treat the disease's symptoms. Levodopa, the drug found in pea seeds, is regarded as the greatest Parkinson's disease treatment currently offered (Yadav *et al*., 2017). Levodopa alone may not be the only situation in which Mp is significant. Therefore, additional research is needed to examine the roles played by other Mp seed constituents in Parkinson's disease. Supplementing with Mp seed enhances locomotor behaviour, according to research conducted on both human beings and animal models (Dhanasekaran *et al*., 2008). Research demonstrates that Mp's antioxidative and metal-chelating properties help to reduce oxidative stress, which in turn improves the redox state. Furthermore, in dROSophila and mouse models of Parkinson's disease, Mp seed extract enhances TH expression and synaptic activities that are essential for neuronal survival. In addition, studies show that Mp seed powder has a quick start to action that lasts a long time without increasing its negative effects. In order to manage PD, Mp seed extract appears to be more beneficial than synthetic levodopa medication (Yadav *et al*., 2013). That said, this benefit might be attributable to another ingredient found in Mp seeds that helps fight the illness. Future research in this environment will therefore improve our understanding. Furthermore, when comparing BME's effects in the PD mouse model, it proves to be more beneficial than Mp seed extract (Yadav *et al*., 2017). But more study on the subject will clarify how the herbal medications compare in terms of their potential effectiveness in treating PD.

2.2 Turmeric: *Curcuma longa* (L.)

Turmeric, *C. longa* (Cl), is a herb that grows forever, its rhizome has been traditionally used for a long time to treat sprains and edema caused by injuries. Numerous studies have demonstrated the great medical usefulness of Cl, which is demonstrated by its anti-depressant, anti-inflammatory, antibacterial, antioxidative, and anticarcinogenic nature. Additionally, Cl protects the brain from ageing, neuronal loss, behavioral abnormalities, and disruption to the blood-brain barrier (BBB). Moreover, studies on neurodegenerative diseases including PD and AD have examined its effectiveness. Research has indicated that curcumin, a crucial constituent of Cl, has antioxidant properties and elevates striatal dopamine levels in PD-affected rats. Moreover, curcumin therapy appears to mitigate the effects of GSH depletion, protecting proteins from oxidation, in DA-ergic brain cells and a mouse model (Zbarsky *et al*., 2005). While maintaining mitochondrial complex I activity, it also guards preventing *in vitro* brain mitochondrial damage and nitrosative stress. In another study, curcumin has shown to protect against neuronal abnormalities in the SN area of the brain using a rat model of PD caused by 6-OHDA. While decreasing MDA levels, curcumin increases SOD and GPx. There was also an observation of up-regulated levels of DA and acetylcholine (Ach). It was also discovered that memory function had significantly enhanced. Another recent study demonstrated that PQ exposed to PINK1 siRNA cells after receiving a curcumin pre-treatment showed enhanced mitochondrial function, reduced apoptosis, respiration, ATP synthesis, and restoration of the mitochondrial membrane potential (Ammon and Wahl, 1991). Thus, curcumin offers significant potential for treating PD. Furthermore, it was demonstrated that curcumin-glucoside, a synthetic curcumin derivative, has the capacity to bind the oligomeric form of a-synuclein, preventing the protein from further fibrillating. This is a significant discovery since it has helped with the creation of drugs that alter disease and may be effective in treating PD. Curcumin also exhibits little toxicity, which is another benefit of using it for PD. Thus, it appears from the above-described results that Cl has a great deal of promise for use as a candidate medication in clinical trials related to PD.

2.3 Green tea: *Camellia sinensis* (L.) Kuntze

Camellia sinensis (L.) Kuntze (Cs) is popular for its health benefits, green tea is produced from dried and steamed Cs leaves. Polyphenols with neuroprotective, anti-inflammatory, anticarcinogenic, anti-oxiDAtive, and antimicrobial qualities are found in Cs. Supplementing with Cs reduces PD risk, according to research (Sharangi, 2009).

Green tea's primary component is catechins, and research has shown that the antioxidative and iron-chelating properties of green tea concentrate, specifically epigallocatechin-3-gallate (EGCG), offer neuroprotection in the mouse PD model generated by 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP). Additionally, it was discovered that adding Cs polyphenols to a 6-OHDA-induced PD model in rats tends to enhance the redox state, which blocks the ROS-NO pathway (Chan *et al.,* 2011). The process involves preserving the ability to scavenge free radicals, which in turn shields the DA-ergic neurons in the striatum and midbrain. A different study revealed that mice's DA-ergic neurons are shielded from MPP+ toxicity by polyphenols found in Cs. It has been demonstrated that it inhibits the DA transporter (DAT), preventing MPP+ absorption through DAT. Additionally, it was discovered that in the MPTP-induced rat model of PD, EGCG from Cs blocked inducible nitric oxide synthase (iNOS). New research shows that EGCG protects DA-ergic neurons from MPTP toxicity in a mouse model of PD. Moreover, it lessens oxiDAtive stress and decreases iron levels in the brain's nigral area. It is clear from this Cs's neuroprotective qualities appear to be advantageous in PD. Future research on its mechanism will therefore give it a stronger founDAtion to be applied as a possible treatment approach against PD.

2.4 Maidenhair tree: *Gingko biloba* (L.)

Ancient Chinese herbals list *G. biloba* (Gb) seeds as having medicinal benefits. It is well recognized that Gb possesses anti-inflammatory, antiageing, antioxidative, and neuroprotective qualities. Research has demonstrated that in Parkinson's disease animal models, Gb extract or EGb 761, provides neuroprotection. According to the study, EGb 761 lowers behavioral abnormalities in rat model of PD induced by 6-OHDA. Moreover, locomotor activity, muscular coordination, and behavioral rotation all significantly improve in the 6-OHDA-induced rat model of PD when Gb supplementation is used (Kim *et al*., 2004). Furthermore, the activity of the GSH-dependent enzymes catalase and superoxide dismutases (SOD) as well as the up-regulation of glutathione (GSH) levels, improved and normalized antioxiDAnt activity. Furthermore, after receiving Gb therapy, the amount of DA and its metabolites dramatically increased, indicating a possible use for Gb in PD treatments (Yang *et al*., 2001). In a different study, it was discovered that Gb reduces oxidative stress and apoptosis in an MPTP-induced rat model of PD by increasing DA levels and SOD activity and reducing malondialdehyde (MDA) levels. Supplementing EGb 761 to a rat model of PD induced by serpine seems to stabilize the redox status, enhance mitochondrial function and ATP generation, and decrease apoptosis (Zhou *et al*., 2017). Additionally, a study demonstrated that Gb extract offers defence against the harmful effects of levodopa that are used during PD treatment. As a result, levodopa with Gb combined in a dose-optimized way may have more therapeutic efficacy than either medication alone. Thus, it would appear that EGb 761, a Gb extract, not only helps to restore mitochondrial function and motor activity in PD but also tends to stabilize the redox status in the disease. Furthermore, EGb 761 can pass acROSs the BBB and has a low molecular weight. Therefore, Gb appears to be a good option for use in PD therapies; nevertheless, preliminary clinical trials are needed to confirm its effectiveness.

2.5 Brahmi: *Bacopa monnieri* (L.) Pennell

A perennial creeping herb with many medical uses is called *B. monnieri*, or Brahmi (Bm). It has been demonstrated to possess antioxidative, anti-inflammatory, antimicrobial, neuroprotective, and memory-boosting qualities. Additionally, Bm extract (BME) is recognised to improve cognitive abilities. Moreover, research has demonstrated that BME has an anti-Parkinsonian impact in transgenic and toxin-induced animal model systems, indicating that it may be effective in treating PD. BME has been demonstrated to protect against paraquat (PQ) toxin in this environment by reducing enhanced oxidative stress in Drosophila and mice (Hosamani *et al.,* 2016). Comparable outcomes have been observed in Drosophila treated with rotenone and in a PD mice model (Shinomol *et al*., 2012). Additionally, BME supplementation in the transgenic PD model of Drosophila reduced apoptosis and oxidative stress. Additionally, in a transgenic PD model in *C. elegans*, BME supplementation reduces a-synuclein aggregation and slows down DA-ergic neuron degeneration. Furthermore, BME therapy lessens the DA-ergic neurons' rotenone-induced cytotoxicity (Siddique *et al*., 2014). By boosting the activity of antioxidative enzymes and improving redox status, it maximizes the normalization of oxidative stress. The data shows that BME supplementation reduces the levels of oxiDAtive indicators such as MDA, hydrogen peroxide, and protein carbonyl content to further support this. Also, BME supplementation enhances mitochondrial function by bringing electron transport chain (ETC) complexes back to normal action. Furthermore, DA-ergic cell lines intoxicated with PQ and 1-methyl-4-phenyl-pyridinium iodide (MPP+), BME also helps to sustain mitochondrial complex I activity and mitochondrial membrane potential (Cm). Moreover, BME promotes the expression of the neurogenic gene in the SN area of the brain and increases tyrosine hydroxylase (TH) activity. It enhances cognitive performance and locomotor activity in PD animal models. In animal models of PD, it enhances cognitive performance and locomotor activity. Consequently, it is abunDAntly clear that BME appears to have enormous promise as a herbal medication for PD. Nevertheless, further research is required to fully comprehend the mechanism of action before it can be used as a possible therapeutic target to treat PD.

2.6 Ashwagandha: *Withania somnifera* (L.) Dunal

W. somnifera (Ws), also known as ashwagandha, is a significant medicinal plant that has been utilized for medical purposes in India since ancient times. Given its aphrodisiac properties and potential as a nerve tonic that improves memory and learning, Ws has a great deal of therapeutic potential (Ahmad *et al*., 2005). Ws roots have antidepressant, anti-inflammatory, antioxidant, memory-boosting and anticarcinogenic, qualities (Prakash *et al*., 2014). They are also anxiolytic. These demonstrate its effectiveness in treating several illnesses, including PD. Research indicates that Ws root extract increases GSH and glutathione peroxidase (GPx) levels, which tends to restore oxidative stress in MPTP-induced mice models of PD (Prakash *et al*., 2014). In addition, it seems to enhance motor function in the Ws-treated animal model of PD and raise dopamine levels in the striatum. A study employing a rat model of PD produced by 6-hydroxydopamine (6-OHDA) found that Ws extract enhances TH expression and restores antioxidant levels, hence reducing oxidative stress (Rai *et al*., 2016). According to a different Maneb-PQ investigation on PD-affected mice, ethanolic extract of Ws reduces iNOS expression and enhances mouse locomotor performance (Shivamurthy *et al.,* 2016). Consequently, research using PD models in mice and rats amply illustrates Ws's capacity to prevent PD. Studies on the Drosophila model of PD are conflicting, in contrast to

the rat or mouse model, since some studies indicate that supplementing with Ws extract is ineffective in recovering the PD phenotype (Manjunath and Murlidhra, 2015). On the other hand, Ws delivery to a dROSophila model recovers the PD phenotype, as demonstrated by other researchers. Therefore, additional research is needed to fully understand Ws' potential in the Drosophila model of PD. Additionally, after Ws treatment, mitochondrial activity significantly increases, enhancing its ability to prevent PD. Furthermore, DA-ergic neuron loss is a characteristic of the PD, thus the rate of apoptosis ought to return to normal. In this environment, Ws treatment to the PD maneb-PQ mouse model results in upregulation of Bcl-2 expression and downregulation of Bax, which controls apoptosis. As a result, Ws appears to provide a solid platform for medication development against PD; nevertheless, additional research is needed to fully confirm its promise.

2.7 Black tea: *Camellia sinensis* (L.) Kuntze

Camellia sinensis (L.) Kuntze (Cs) is a popularly eaten oxidized form of tea produced by fermentation is called black tea. It has a therapeutic effect since it contains flavin. The antioxidant properties of tea leaf extract are comparable to those of green tea catechins. Research has demonstrated the antioxidant and neuroprotective qualities of black tea. Black tea protected against 6-OHDA activation of neurons in a rat model of PD, as evidenced by increased tyrosine hydroxylase (TH) expression in the substantia nigra (SN) area of the brain. Furthermore, it was found that supplementing with black tea before receiving 6-OHDA treatment had a greater neuroprotective impact (Chen *et al*., 2011). The benefit is attributed to theaflavin, a multimeric polyphenol present in black tea, albeit the precise neuroprotective mechanism is unclear. It is also recognized to have the strongest catechins in Cs. Additionally, it has been demonstrated that theaflavin enhances the expression of TH and dopamine transporter (DAT) in MPTP-intoxicated C57BL/6 mice's nigral brain region. Additionally, it was noted that theaflavin had an antiapoptotic impact in the Parkinson's disease-induced MPTP-mice model, as evidenced by the decreased expression of caspase 3, 8, and 9. Recent research has shown that introducing black tea to a transgenic Drosophila PD model, which expresses human a-synuclein, raises the DA level and reduces oxidative stress in the flies. Thus, it appears that black tea, like green tea, has good neuroprotective potential. However, a Chinese health research team in Singapore discovered that while green tea does not reduce the incidence of PD, consuming black tea does. Studies that draw comparisons between black and green tea are required to comprehend the underlying molecular mechanisms of action. Moreover, black tea contains chlorogenic acid, which has been demonstrated to raise human total plasma homocysteine (Hcy) levels (Ravindranath *et al.,* 2006). This is crucial because PD patients have higher Hcy levels, which may have an impact on mitochondria-mediated neuronal cell death. As a result, future research on black tea supplementation needs to be carefully evaluated.

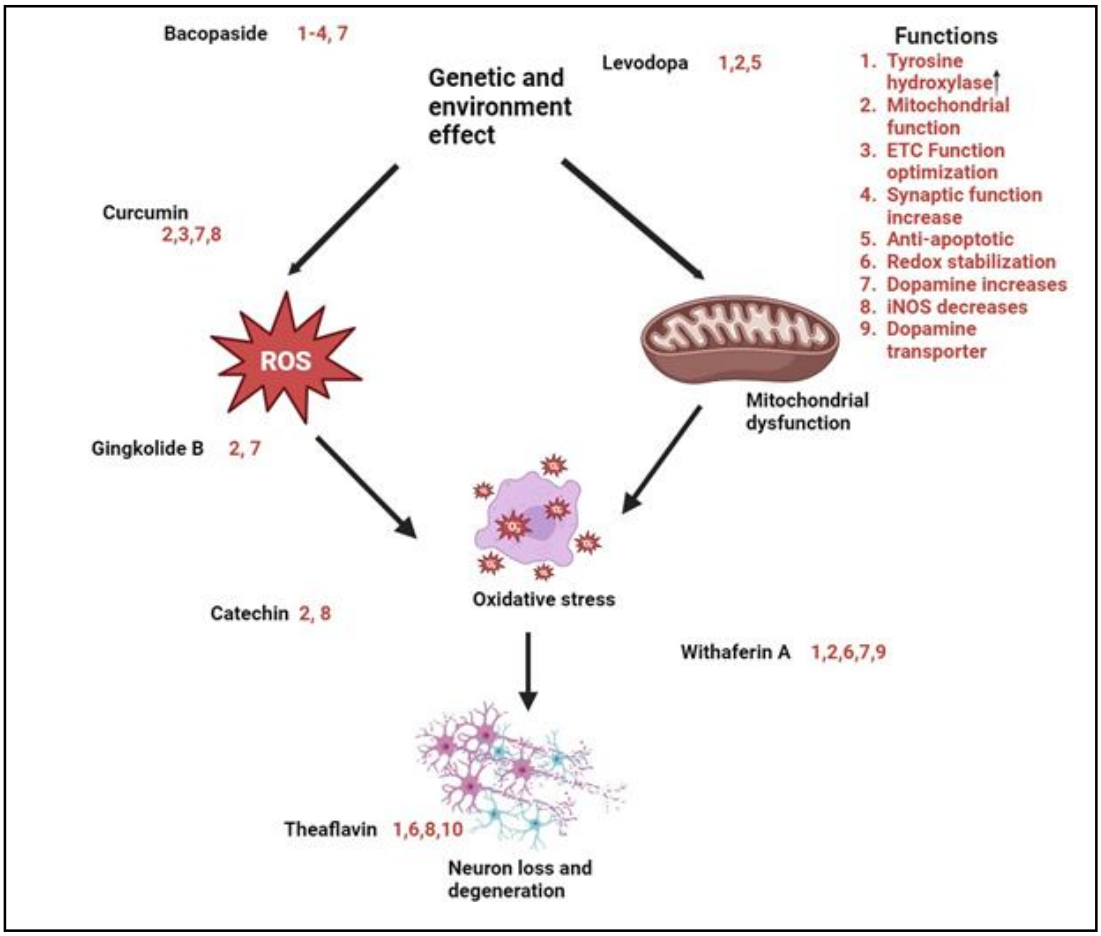

Figure 1: Above image shows the target action of different plant bioactive against Parkinson's disease.

3. Conclusion

Only a few plants, nevertheless, were chosen for this review due to their high anti-inflammatory and antioxidant properties combined with low toxicity. A somewhat similar mechanism of action involving redox stabilization and mitochondrial activity replenishment was discovered to be shared by all plants. This is especially significant because oxidative stress and mitochondrial dysfunction are thought to play a major role in the pathophysiology of Parkinson's disease. All of the plants mentioned, nevertheless, have specific components that have beneficial effects with low toxicity. Additionally, it was found that they offer comparable advantages in sporadic (toxin-induced, such as MPTP, PQ, Maneb, and Rotenone) and familial (transgenic animal model or human patient) forms of Parkinson's disease. Furthermore, as demonstrated by the use of Mp seed extract in therapy, natural herbs offer superior protection as compared to levodopa treatment. Additionally, the potential effectiveness of these plants differs because BME was discovered to have a higher level of neuroprotection than Mp seed extract. These results point to the necessity of comparing these plants' potential therapeutic uses. Information regarding medical importance, toxicity, and effective dosage will be provided by this method. Also, it was found that these herbal extracts improve mitochondrial activity, which shields DA-ergic neurons in the brain's SN region. To investigate the underlying molecular mechanism and related signaling pathways, more research is necessary. In summary, it appears that natural herbs have a great deal of promise, and further research is needed to determine how they work in order to provide a fresh approach to treating this crippling condition.

Conflicts of interest

The authors declare no conflicts of interest relevant to this article.

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