

# A Review on the Relationship between Parkinson Disease and Toxoplasmosis

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## ABSTRACT

Parkinson's disease is considered a progressively progressive disorder that affects the nervous system and many parts of the body that controlled by nerves. Symptoms begin appear as slowly. The first symptom that appear is a tremor occur in only one hand. The symptoms of Parkinson's disease unfortunately get worse as your condition worsens over time. Parkinson's disease causes some nerve cells in the brain to gradually become damaged or die. This is due to the loss of nerve cells that produce the chemical transmitter in the brain called dopamine, the decline of which causes irregular activity in the brain, leading to movement problems in addition to other symptoms of Parkinson's disease. Toxoplasma gondii is a protozoan intracellular parasite when the cyst or bradyzoite stages are present in the brain cells begin to manipulate the normal behavior of the infected person, leading to the appearance of neurological and psychological symptoms in the infected people. there are many studies conducted on human and animal models that have concluded that there is a relationship between infection with toxoplasmosis and the psychological and neurological disorders that affect the carrier of the infection, and this is due to the parasite's ability to infect nerve cells and glial cells, which leads to the death of neurons, the release of inflammatory cytokines, and also causes neurochemical changes as dopamine signaling in the central nervous system changes. Several epidemiological and experimental studies have investigated the role of Toxoplasma gondii infection in the incidence and severity of Parkinson's disease. We designed review to evaluate the relationship between T. gondii infection and the occurrence of Parkinson's disease so that this study would be useful for better understanding the risk factors for neurological disorders.

**Key Words:** Dopamine, parkinson disease, toxoplasmosis, toxoplasma gondii.

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## INTRODUCTION

One of the most common diseases in the world is Parkinson's disease, which is considered one of the common neurological disorders<sup>[1,2]</sup>, as those afflicted with this disease suffer from motor signs which include bradykinesia, rigidity, postural instability, and tremors, and non-motor dysfunction which include disturbances of sleep, sensory untidiness, disturbances of mood, and cognitive impairment<sup>[3-6]</sup>, and the life span of people afflicted with this disease is short<sup>[7]</sup>, as There is a threat of death the longer the illness lasts, and the patient also suffers from poor quality of life<sup>[8,9]</sup> The causes of this disease are not yet clear, and it threatens society as a cause of death, but some implicated include neuroinflammation,<sup>[10, 11]</sup> dysfunction of mitochondria,<sup>[12-14]</sup> oxidative stress,<sup>[15]</sup> and homeostasis of defective protein, play a role in infection<sup>[16, 17]</sup>. so it is

recommended to focus as much as possible on preventing this disease<sup>[18]</sup>.

Parkinson s disease occurs after the mislaying neurons of dopamine in the pars compacta of the substantia nigra, related to the existence of an intracellular collection of misfolded alpha-synuclein<sup>[5,6]</sup>.

The Toxoplasma parasite is an obligate parasite that infects warm-blooded animals, including humans. Cats represent their only host, as they shed eggs in their feces, causing environmental pollution<sup>[19-21]</sup>. The infection is transmitted to humans by consuming water contaminated with these eggs or eating meat that contains tissue cysts and is not well cooked<sup>[22]</sup>. The infection may be asymptomatic if the person has high immunity, and sometimes it causes

lymph node disease, followed by a latent phase throughout life, but in most cases the parasite becomes active in the case of weak immunity<sup>[23]</sup>, causing encephalitis and psychological and behavioral disorders. The infection also The *Toxoplasma* parasite can cause birth defects in the newborn if it is transmitted from the mother to the fetus<sup>[24-29]</sup>.

Toxoplasmosis is possibly linked with different neuropsychiatric dysfunctions. toxoplasmosis can affect many regions in the central nervous system such as the cerebral hemisphere, basal ganglia, cerebellum, and brain stem respectively. The *Toxoplasma* parasite is one of the obligate parasites that infect humans as a result of consuming water contaminated with mature parasite eggs or eating undercooked meat. It can also be transmitted vertically<sup>[30]</sup> so in this review, we aimed to study the possible association between Toxoplasmosis and Parkinson s disease.

### **Relationship between Toxoplasmosis and Nervous system**

The *Toxoplasma* is a parasite that infects animals and causes zoonotic diseases in humans. The infection is mild in healthy people and may cause death in people who suffer from immunodeficiency as a result of the reactivation of the infection in the central nervous system. If the infection is of the acute type, the severity of the symptoms of the disease will be reduced by the immune response based on interferon, which determines the spread of the parasite. However, if the parasite spreads will differentiate into semi-dormant cysts that form inside nerve cells and muscle cells, thus remaining for life, as the immune response works in the central nervous system to control the injury balance and thus reduce the severity of the injury and the damage resulting from it<sup>[31]</sup>.

Some experimental evidence suggests that the parasite enters the brain via brain endothelial cells or by using dendritic immune cells<sup>[32]</sup>. *Yin et al.*,2022 found that the amount of dopamine in the cortex region of the brain of mice infected with the *T. gondii* parasite is higher than its quantity in the control group of mice, while the infection led to a decrease in the levels of serotonin and norepinephrine in the cortex and amygdala regions of the same mice compared to healthy mice<sup>[33]</sup>. determined the presence of cystic stages of the *Toxoplasma* parasite exclusively in nerve cells and the absence of infection in astrocytes in the brain during chronic infection in mice using dye name FITC-Dolichos biflorans dye and examining them with confocal a special type of microscope named fluorescence microscope<sup>[34]</sup>.The parasite's tendency to reside in the

brain or specific areas therein is one of the hypothesized mechanisms for causing behavioral changes in its hosts<sup>[35]</sup>.

The study of *Di Nardo et al.* (2000) confirmed Profilin is a protein essential for the spatial and transient control of the growth of actin microfilaments, which has a crucial role in controlling the rapid change of cell shape and organ development, wound healing, and the pursuit of invading hostile bodies by cells of the immune system<sup>[36]</sup>.

Al-Tamimi's (2017) study showed that serum levels of human profilin-1 were lower in people with positive IgG and IgM antibodies to the *T. gondii* parasite compared to healthy people, and attributed the reason for the decrease in profilin levels upon infection. Acute and chronic infection with the parasite indicates that the parasite may consume this protein in its effectiveness and activity inside the cells, as it may harness it in the process of its entry or invasion of cells, since profilin is necessary for cellular growth, changing cellular shape, and repairing damaged tissues, or its decrease may be due to its consumption through immune activity formed against the infection. With the *Toxoplasma* parasite, the effect of the parasite on the level of profilin (decrease) may be one of the mechanisms by which the parasite causes neurological symptoms<sup>[37]</sup>.

### **Parkinson's disease**

The disease results from the decomposition of dopamine-generating neurons in the brain. Clinical signs of the disease include mental regression, cognitive decline, and other non-motor symptoms, then move to motor symptoms, which are external movements that appear clear. on the body<sup>[38]</sup>.

When the dopamine-generating region in the brain within the pars compacta of the substantia nigra, which is the precursor to neurodegeneration, is lost or dies, clinical symptoms begin to appear, the most common of which are bradykinesia, rigidity, and tremor<sup>[39]</sup>.

This disease is a progressive disease that affects the functioning of the nervous system and leads to disruption in the movement of the affected person. The symptoms grow gradually. The symptoms of the disease may begin with the trembling of only one hand in the affected person. Although tremor is the most well-known sign of the disease, there are common symptoms. Others, such as stiffness and slowed body movement, and the affected person's face appears devoid of expressions, the arms do not swing when walking, and there is a change in the tone

of the voice when speaking. The symptoms and disease condition increase with time, and although there is no treatment for the disease, taking medication improves the symptoms of the disease and may Doctors suggest brain surgery to improve symptoms<sup>[40]</sup>.

Accurate epidemiological information is necessary to determine the factors that help and enable the identification of Parkinson's disease, plan the provision of health services, and set priorities for conducting medical research on it. The two most important risk factors that are related to the disease are advanced age and family history (genetic factors)<sup>[41]</sup>.

Statistics show that an estimated one million Americans have Parkinson's disease, with 60,000 new cases diagnosed each year. The total number of people suffering from the disease around the world is about 7-10 million, and its prevalence is expected to double for people over the age of 50 by 2030<sup>[42]</sup>.

The increase in cases of Parkinson's disease is linked to the increase in the world's population (*Dorsey et al.*, 2007), and the continuation of the current trend of population aging, and this may appear clearly in the next two decades. The minimum incidence of the disease has been estimated at 22.8/100,000 each year in Europe and the United States. United States of America<sup>[43]</sup>.

Predictions and studies indicate that the incidence of Parkinson's disease will increase in people aged 60 and over by about 6 million people between 2009 and 2030 in Russia. (*Costa et al.*, 2010) Cases range from 9 per 100,000 in Eastern Europe to 22 per 100,000 in the city of Aberdeen in eastern Scotland, with a similar annual rate in the Newcastle-Gateshead area of Britain of 15.9/100,000<sup>[44]</sup>.

Parkinsonism includes five movement symptoms including: tremor, rigidity, and slowness of movement, as well as postural instability (balance problems) and difficulty walking<sup>[45]</sup>. The first four symptoms are major, namely tremor, rigidity, slow movement, and instability, while the last are secondary<sup>[46]</sup>. Tremor is the main clear symptom of the disease, which means spontaneous, rhythmic and oscillatory actions of the body parts. This symptom usually appears during a state of rest and may also occur during the patient's postural state of movement<sup>[47]</sup>. The second symptom is stiffness that may occur in any part of the body (muscle stiffness). Stiff muscles can limit the range of motion and cause pain for the patient. The symptoms of Parkinson's disease are the beginning of an asymmetrical distribution of nerve impulses. During the disease, the symptoms of Parkinson's disease are a gradual and

progressive development of its stages, and some cases of asymmetry usually persist. Manifestations of movements include disturbances in the patient's posture, for example, reduced arm extension while walking, as well as the use of short steps and difficulty speaking and swallowing<sup>[48]</sup>. The fourth main symptom is slowness of movement. At this stage of the disease, the patient's ability to move may be reduced or his movement may slow down, and his steps may become short and difficult to walk<sup>[49]</sup>. In addition to other manifestations such as a mask-like facial appearance or short handwriting, it is typical of the range of motor distortions that can appear. In specialized centers, the accuracy of diagnosis can be higher, as many diagnostic criteria are adopted to increase the accuracy of diagnosing paralysis<sup>[50]</sup>.

Diagnosis requires the presence of two of the four basic symptoms (in terms of appearance). Imaging techniques, such as positron emission tomography, single-photon emission tomography, and magnetic resonance imaging (MRI), are also included in the diagnosis, which can help to some extent. What distinguishes Parkinson's disease from other similar diseases<sup>[51]</sup>.

Parkinson's disease itself does not cause death, but symptoms related to the disease can be fatal. For example, injuries caused by falls or problems associated with dementia can be fatal, and some people with the disease may have difficulty swallowing, which can lead to pneumonia<sup>[52]</sup>.

Non-motor manifestations include cognitive changes such as difficulty with memory or planning, slowed thinking as well as mood disorders such as anxiety and depression, sleep disturbances such as insomnia, fatigue, constipation, and vision problems. Symptoms of dizziness may require additional treatment in many people and these symptoms can develop as the disease progresses<sup>[53]</sup>.

### Causative Agents of Parkinson

The cause of Parkinson disease is still unknown, although there are some indications may lead to this disease such as genetics and environmental factor, or it may be a combination of the two. It is also it thought that there are more than one cause of the disease, as scientists believe that both genes and the environment interact and lead to cause Parkinson's disease<sup>[54]</sup>.

Some scientists have found clumps of certain substances (protein) inside brain cells called Lewy bodies in affected people. It is believed that this protein is related

to Parkinson's disease, as these proteins were found to accumulate in the brain, the production of Dopamine may cause programmed cell death<sup>[55]</sup>.

Some studies have linked the disease to chronic exposure to certain substances, such as heavy metals, pesticides, and toxins in particular. Epidemiological studies show an increased prevalence of Parkinson's disease in people who are constantly exposed to pesticides and herbicides, especially paraquat (a non-selective herbicide that has been used for many years in agriculture). In most countries of the world, researchers have found that chronic exposure to paraquat leads to a loss of dopamine, and although it is not clear why these changes occur, there are some reports that indicate that exposure to certain toxins or environmental factors may increase the risk of the disease<sup>[56]</sup>.

Some neurons in the brain are damaged or die gradually (physiologically), as this indicator is due to the loss of dopamine-forming neurons. When the amount of dopamine decreases, it leads to abnormal work in the brain and muscles, leading to the appearance of symptoms of Parkinson's disease<sup>[57]</sup>.

### 1. Genetic agents

Scientists have discovered specific genetic mutations that can lead to Parkinson's disease, but these cases are rare and occur in some families affected by the disease. Despite this, most cases of Parkinson's disease remain, and no underlying genetic cause can be identified<sup>[58]</sup>. Parkinson's disease is largely associated with the occurrence of numerous and varied mutations in some genes related to the disease, such as the LRRK2 gene (leucine-rich repeat kinase group). The defect has been found to be frequent in some families that are afflicted with the disease, as well as in North African or Jewish families<sup>[59]</sup>.

Many studies have recorded the occurrence of mutations in many genes related to this disease, including the genes SNCA,  $\alpha$ -synuclein, LRRK2, PRKN, PINK1, and DJ-1, all of which contribute to damage to mitochondria within cells and their association with the disease<sup>[60]</sup>.

### 2. Environmental agents

Most research has reported a consistent increase in the risk of developing Parkinson's disease with exposure to environmental factors such as rural living and agriculture, such as the use of some types of pesticides and fertilizers<sup>[61]</sup>.

Many if the researchers have also found many changes that occur in the brains of people have Parkinson's disease, although there is no clear reason for these changes to occur, but some reports indicate that exposure to certain toxins or environmental factors may increase the risk of the disease occurring in later, but the risk is relatively small<sup>[62]</sup>.

### 3. Pathological agents

Poskanzer and Schwab established their hypothesis known as the Poskanzer and Schwab hypothesis (PSH), in which they attributed Parkinson's disease to infection with influenza in the 1950s. Since that time, this hypothesis has prompted researchers to draw their attention and interest in linking infections with pathogenic causes with Parkinson's disease. Despite Although the proof of the original hypothesis has not been completed, it has received many opinions, acceptance and rejection by researchers. A feasibility study for quantitative assessment of cerebrovascular malformations using flutriciclamide<sup>[63]</sup>.

Many studies indicate that there is a relationship between Parkinson's disease and some pathogens. Some studies have indicated that there is a relationship between the presence of *Helicobacter pylori* bacteria in the stomach and Parkinson's disease. Although the exact mechanism is not known precisely, an increased prevalence of stomach ulcers has been observed in patients. Parkinson's disease, which was initially thought to be a gastrointestinal symptom associated with Parkinson's disease, until researchers found the infectious *Helicobacter pylori* bacteria, which is believed to be a relationship with the disease<sup>[64]</sup>.

Some proposed hypotheses have explained why this bacteria causes Parkinson's disease. They are that the bacteria show high toxicity that affects the nervous system by increasing glucosides and cholesterol, as well as by deteriorating dopaminergic nerve cells in the brain. It is believed that the bacteria release chemicals that help in excessive activation of parts of the brain that lead to brain damage<sup>[65]</sup>.

There are other hypotheses that explain that the human immune system cannot eliminate bacteria, which leads to the development of Parkinson's disease by damaging dopamine cells in the brain<sup>[66]</sup>.

Some researchers have attributed the relationship between this bacteria and Parkinson's disease by causing programmed death of nerve cells after crossing the blood-brain barrier after being swallowed orally or inhaled by the nose<sup>[67]</sup>.

Viruses have a role in participating in the factors that cause the disease, as it was found that the hepatitis C virus, type HCV, may invade the central nervous system after infecting the liver, and that both HCV and Parkinson's disease share the same damage to the nerves and the same inflammatory biomarkers, as the HCV virus causes the death of nerve cells. Dopaminergic activity increased by 60% in the glial neuron organoid culture system in mice. This finding was further supported by the finding that HCV infection releases inflammatory cytokines, which may play a role in the pathogenesis of Parkinson's disease<sup>[68]</sup>.

Parasitic infections play a major role in neurological diseases, including Parkinson's disease, as Park (2017) recorded the appearance of symptoms of Parkinson's disease in a patient infected with the malaria parasite *Plasmodium vivax* in South Korea, and many studies recorded a link between Parkinson's disease and infection with the parasite *T. gondii*<sup>[69]</sup>.

## Dopamine

It is an organic chemical of the catechol-amine and phenethyl-amine family that plays in the body and specially many important roles. It is an amine that is produced from the amino acids phenylalanine and tyrosine. The first amino acid is converted to the second by the enzyme Phenylalanine hydroxylase and by the action of two other enzymes, Tyrosine hydroxylase and Dopa decarboxylase, through two new steps. Dopamine is finally produced, despite the availability of both phenylalanine acids. And tyrosine in the brain, but the starting point for the formation of dopamine in the brain is through the amino acid tyrosine, and although the concentration of tyrosine is high in the brain, it is used at a rate of 1% in the production of dopamine, and despite the presence of phenylamine in the brain, it is not used in the production of dopamine, but rather it depends on the conversion of phenylamine present in the liver into tyrosine, which is then transported to the brain and converted into dopamine<sup>[70]</sup>.

Dopamine, which is secreted from nerve cells, is a neurotransmitter that transmits nerve signals from one cell to another or from one nerve cell to the muscles<sup>[71]</sup>. These nerve cells are stimulated through several pathways located within the behavioral stimulation area of the brain<sup>[72]</sup>.

In 1910, dopamine was discovered, as it was diagnosed as a chemical substance that helps in the production of another chemical substance called norepinephrine, but it was found that dopamine is affected by many factors that increase the amount of its secretion or hinder its absorption by nerve cells. Dopamine is unable to cross the blood-brain

barrier and its function is limited to the peripheral areas independent of the brain, where it controls the release of hormones and other activities<sup>[73]</sup>.

There are studies that have shown that dopamine is present in the bloodstream in large quantities without knowing its importance and function there. It was found that blood plasma contains a large percentage of dopamine, which is present in the form of dopamine sulfate, and its percentage was compared with the percentage of adrenaline, as the percentages were similar between them. The mesentery in the digestive system contains large amounts of dopamine sulfate, as it works to remove dopamine toxins that are taken orally or that are secreted during the digestion process. The percentage of dopamine also increases after each meal, and this increase was measured in the blood plasma and is excreted in the urine and has many biological functions<sup>[74]</sup>. A small percentage of dopamine may penetrate the blood-brain barrier and be present in the bloodstream, and the sympathetic nervous system and the digestive system may produce a small amount of free dopamine, which is received by the walls of the arteries due to the presence of dopamine receptors on the surfaces of these walls, where it expands the blood vessels and inhibits norepinephrine<sup>[75]</sup>.

Dopamine also binds to specific receptors on immune system cells such as lymphocytes, so it plays an important role in influencing the immune system by affecting immune cells in peripheral systems such as the circulatory system, bone marrow, and spleen. These immune cells there are able to manufacture dopamine and release it into the blood, where it modulates blood flow<sup>[76-79]</sup>. Dopamine reduces the activity of lymphocytes, which may lead to autoimmune diseases, and therefore it plays an important role in the interactions between the nervous system and the immune system<sup>[80]</sup>.

There are also receptors for dopamine on the nephron cells, where tubular cells manufacture and release it in the kidney, which increases blood flow, thus increasing the glomerular filtration rate and increasing sodium secretion in the urine. This means that a decrease in the sodium level may occur when there is a malfunction in the dopamine in the kidneys, so the body's blood pressure rises. High blood pressure occurs when there is a malfunction in the renal dopamine system for reasons that may be genetic due to increased oxidative stress due to the production of dopamine or a malfunction in the receptors on the nephron cells, which causes high blood pressure<sup>[81]</sup>.

The exocrine cells of the pancreas can also produce dopamine because they have dopamine receptors on their surface. Previous research has found that insulin-secreting

beta cells can also make dopamine, which in turn reduces the amount of insulin secreted. Dopamine may come from other cells in the pancreas, such as cells of the sympathetic nervous system<sup>[82]</sup>.

Dopamine protects the intestinal mucosa from damage, as dopamine reaching the small intestine limits the movement of the digestive system. Therefore, the function of dopamine varies from one place to another, depending on its location in the different cells and organs of the body<sup>[83]</sup>.

Dopamine is available in pharmacies under different and varied names depending on the manufacturing companies. The most common names that are listed by the World Health Organization as essential drugs used as a stimulant and regulator of heart rate and blood pressure disorders. It may be used specifically as a treatment for newborns. These most common trade names are Intropin, Dopastat, and Revimine<sup>[84]</sup>. When used as a treatment for newborns, it is given via intravenous drip more than once. Its half-life in plasma is two minutes, and its half-life for premature babies is five minutes. As for adults, its half-life is very short, which is only one minute<sup>[85]</sup>.

A malfunction may occur in the dopamine-producing cells when concussions occur in the brain due to sports or due to chemical poisoning (MPTP). These reasons may lead to damage to these dopamine-producing cells and activation of programmed cell death, which leads to many diseases, the most important of which is Parkinson's disease<sup>[86]</sup>.

The study of *Muhammad et al.* In 2022, which was conducted on mice, the level of dopamine in the cerebrospinal fluid and serum increased in mice infected with the *Toxoplasma* parasite and Parkinson's disease compared to mice infected with Parkinson's disease only, which proved that regulating the increase in the level of dopamine due to infection helps reduce the complications of Parkinson's disease<sup>[87]</sup>.

Researcher Mahami proved in 2016 in Iran that there is no significant relationship between the concentration of *Toxoplasma* antibodies and the incidence of Parkinson's disease, even though there is a significant relationship with  $p = 0.03$  between raising cats and Parkinson's disease, as well as between eating undercooked eggs and Parkinson's disease,  $p = 0.004$  despite Parkinson's patients recorded a high level of IgG, and therefore no relationship between Parkinson's disease and infection with the *Toxoplasma* parasite was recorded<sup>[88]</sup>.

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## CONCLUSIONS

We conclude from the current review that dopamine is considered one of the indicators of Parkinson's disease, as studies have shown that the majority of those infected suffer from low dopamine levels. Infection with the *Toxoplasma* parasite also contributes to the increased spread of this disease, through research that has confirmed the positive relationship between infection with the parasite and the spread of Parkinson's disease, resulting from the effect of the parasite on the cells responsible for secreting dopamine in the brain.

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## RECOMMENDATIONS

1. Holding awareness seminars and courses for women through primary health care units to clarify the relationship between infection with the toxoplasmosis parasite and Parkinson's disease.
2. The necessity of monitoring dopamine levels in women with toxoplasmosis.
3. Future studies interested in finding a definitive cure for Parkinson's disease or reducing it.

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## مراجعة العلاقة بين مرض باركنسون وداء المقوسات

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مرض باركنسون هو اضطراب تقدمي تدريجي يؤثر على الجهاز العصبي وأجزاء الجسم التي تتحكم فيها الأعصاب. تبدأ الأعراض ببطء. العرض الأول هو رعشة في يد واحدة فقط. تزداد أعراض مرض باركنسون سوءاً مع تفاقم حالتك بمرور الوقت. يتسبب مرض باركنسون في تلف بعض الخلايا العصبية في الدماغ تدريجياً أو موتها. ويرجع ذلك إلى فقدان الخلايا العصبية التي تنتج الناقل الكيميائي في الدماغ المسمى الدوبامين، والذي يؤدي تراجعها إلى نشاط غير منتظم في الدماغ، مما يؤدي إلى مشاكل في الحركة بالإضافة إلى أعراض أخرى لمرض باركنسون. التوكسوبلازما جوندي هو طفيل أولي داخل الخلايا عندما تتواجد مراحل الكيس أو البراديزويت في خلايا الدماغ تبدأ في التلاعب بالسلوك الطبيعي للشخص المصاب، مما يؤدي إلى ظهور أعراض عصبية ونفسية لدى المصابين. هناك العديد من الدراسات التي أجريت على نماذج بشرية وحيوانية خلصت إلى أن هناك علاقة بين الإصابة بداء المقوسات والاضطرابات النفسية والعصبية التي تصيب حامل العدوى، ويعود ذلك إلى قدرة الطفيل على إصابة الخلايا العصبية والدبقية، مما يؤدي إلى موت الخلايا العصبية، وإطلاق السيوتوكينات الالتهابية، ويسبب أيضاً تغيرات كيميائية عصبية مع تغير إشارات الدوبامين في الجهاز العصبي المركزي. بحثت العديد من الدراسات الوبائية والتجريبية في دور عدوى التوكسوبلازما جوندي في حدوث وشدة مرض باركنسون. لقد قمنا بتصميم مراجعة لتقييم العلاقة بين عدوى المقوسة الغوندية وحدث مرض باركنسون بحيث تكون هذه الدراسة مفيدة لفهم عوامل الخطر للاضطرابات العصبية بشكل أفضل.