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# MOLECULAR DETECTION AND GENETIC CHARACTERIZATION OF TOXOPLASMA GONDII INFECTION IN PREGNANT WOMANS

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**ABSTRACT:**
Toxoplasmosis is a disease caused by T. gondii, a protozoon which affects humans and animals and is widely distributed worldwide. In humans, there is great concern due to the serious consequences that can infection of pregnant women and the newborn. The early diagnosis of gestational toxoplasmosis is important for occur in the treatment to be carried out in order to prevent vertical transmission or reduce damage and PCR of amniotic fluid. Previous studies have also reported PCR of the placenta as a good diagnostic test. The development of simple, sensitive and rapid methods for the detection and identification of Toxoplasma gondii is important for the diagnosis and epidemiological studies of the zoonotic disease toxoplasmosis. The application of these methods has generated invaluable information to enhance our understanding of the epidemiology, population genetics and phylogeny of T. gondii. However, since most studies focused solely on the detection but not genetic characterization of T. gondii, the information obtained was limited. In this review, we discuss some widely use molecular methods and propose an integrated approach for the detection and identification of T. gondii, in order to generate maximum information for epidemiological, population and phylogenetic studies of this key pathogen.
**Keywords**: Toxoplasma gondii, genetic characterization, molecular detection, pregnant womans.

## INTRODUCTION

Toxoplasma gondii is an obligate intracellular, apicomplexan parasite that infects all warm-blooded vertebrates, including mammals and birds. It is the only known species in the genus Toxoplasma and is considered to be one of the most successful eukaryotic
pathogens in terms of the number of host species and percentage of animals infected worldwide. Up to one-third of the human population in the world is chronically infected (Dubey and Beattie, 1988; Tenter et al. 2000). Toxoplasmosis is disregarded in some countries (Wallon and Peyron,2018) and considered a neglected parasite disease by the Centers for Disease Control and Prevention (CDC). There are still many failures and difficulties in diagnosing toxoplasmosis. Soares and Caldeira (2019) toxoplasma gondii is an obligate intracellular protozoan parasite with a global distribution in humans and other warm-blooded animals. It is a coccidian parasite of cats as final hosts, and all non-feline warm-blooded animals (including humans) as intermediate hosts.1 About one-third of the world's

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human population is infected with the parasite.2Intermediate hosts, including humans, acquire T gondii by ingesting either tissues of infected animals, food and drink contaminated with speculated oocysts from cat feces or soil, tissue cysts contained in undercooked meat or meat byproducts containing the cysts.3,4 Although Toxoplasma infection is often benign, congenital toxoplasmosis can lead to severe sequelae for the fetus and new born.5,6 The infection may cause miscarriage, death

## PATHOGENESIS

Human infections are primarily caused by ingesting undercooked meat containing viable tissue cysts or by ingesting food or water contaminated with oocysts shed in the faces from infected cats (Dubey, 2004). Primary infections in adults are mostly asymptomatic but lymphadenopathy or ocular toxoplasmosis can occur in some patients. Severe acute, disseminated toxoplasmosis can occur in immunocompetent human patients when infected with some isolates (Bossi and Bricaire,2004). Infection acquired during pregnancy may spread and cause severe damage to the foetus. In immune compromised patients, the reactivation of a latent infection can cause life-threatening encephalitis (Montoya and Liesenfeld, 2004)**.** Humans may become infected by ingestion of contaminated food or water, vertical transmission, and more rarely by blood transfusion or organ transplantation of infected persons (Dubey et al., 2012). The infection is often symptom-free or mild, but if it occurs during pregnancy it can have serious consequences for the fetus, like retinochoroiditis, cerebral calcifications, hydrocephalus, abortion, neonatal death (Joiner and Dubremetz, 1993). These verity of the clinical manifestation is related to the gestation period. The consequences for the fetus are more severe in early pregnancy (Cook et al., 2000).fiction occurring later in pregnancy may result in either congenital disease or subclinical infection. The transmission of T. gondii to the fetus is often linked to primary maternal infection during pregnancy. T. gondii infection during pregnancy can result in serious congenital infection, including abortion (Chaudhry S.A., Gad N., Koren G. 2014). Congenital toxoplasmosis is the most serious manifestation of infection resulting from the vertical transmission of T. gondii transplacentally from a parasitic mother to her offspring. These verity of disease depends on the gestational age at transmission Iqbal (J., Khalid N. 2007). In addition, to the trimester of maternal infection was acquired. For untreated women, the rate of transmission is nearly 25%, 54%,65% respectively (Maldonado,2017). However, approximately 50%of frequent miscarriages are not attributed to these etiological factors and their causes remain unknown. However, 13 million HIV-infected people were Toxoplasma-seropositive worldwide, thus at risk for cerebral toxoplasmosis, with 87% of them living in sub-Saharan Africa (Shahine L., Lathi R. 2015). In Arab world including Iraq, toxoplasmosis has high prevalence rate of infection in pregnancy patients (Basavaraju A. 2016). The acute infections in pregnant women can lead to congenital toxoplasmosis, which may cause blindness, mental retardation, or even death of the fetus (Baqer N.N., Saheb E.J., Ahmed N.S. 2021)

## EPIDEMOLOGY

T. gondii has subpopulation structures in different geographical regions. It has what appears to be a clonal population structure in North America and Europe, with 3 predominant lineages (named types I, II and III) defined by multi-locus enzyme electrophoresis (MLEE), polymerase chain reaction restriction fragment length polymorphism (PCRRFLP) or

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microsatellite analysis (Darde et al. 1992; Howe and Sibley, 1992; Ajzenberg et al. 2002a). A recent report suggests the same clonal structure in Africa (Velmurugan et al. 2008). However, T. gondii isolates from animals and human patients in South America are diverse and largely distinct from those in North America and Europe (Ajzenberg et al.2004; Khan et al. 2006; Lehman et al. 2006; Dubey et al. 2008). Historically, these distinct and non-type I, II or III parasites were designated as the 'atypical' or 'exotic' isolates. The low linkage disequilibrium among genetic loci of these isolates suggests that the parasites have undergone frequent sexual recombination (Ajzenberg et al.2004; Lehman et al. 2004; Su et al. 2006). The consequences of infection with T. gondii depend on the host species and parasite genotypes. The prevalence of human T. gondii infection varies in different parts of the world, and has been reported with rates up to 75% (Pappas et al.,2009). Specifically, in Brazil, in pregnant women, there are studies that indicate prevalence of chronic infection from 42% to 90% prevalence of toxoplasmosis is continuingly evolving, subject to regional socioeconomic parameters and population habits. It presents in every country in the world and seropositivity rates range from less than 10 % to over 90 %. In African countries, numerous studies performed in the early 1990s with limited follow-up exists even for the (Al-Toban H.A., Al-Marsomy H.D., Al-TameemiW.F., Al-Obaidi A.B., Mohammed M.A., Al-SaeedR.M., Al-Shemary I.K. 2019). summarized prevalence rates in Egypt general population of these countries. Recent review by (57.9 %), Tunisia (58.4 %), Morocco (50.6 %), Nigeria (20.8 %), Mali (21 %), Benin (3.6 %), Gabon (71.2 %), Madagascar (83.5 %), and Senegal (40.2 %).

## MOLECULAR METHODS FOR THE DETECTION

Molecular methods rely on PCR for the specific detection or analysis of T. gondii DNA. These methods have proved to be simple, sensitive, reproducible and cost-effective, and have been applied to a variety of clinical samples from animals and humans (Belland Ranford-Cartwright, 2002; Contini et al. 2005; Calderaro et al. 2006; Bastien et al. 2007). Molecular methods can be divided into 2 groups. The FirstGroup focuses on specific detection of T. gondii DNA in biological samples. The conventional PCR, nestedPCR (n-PCR) and quantitative real-time PCR(qPCR) of repetitive DNA sequences belong to this group. The second group of molecular methods focuses on a high resolution identification of T. gondii isolates. The multilocus PCR-RFLP, microsatellite, and multilocus sequence typing (MLST) of single copy DNA sequences belong to this group. Molecular detection by conventional PCR, nestedPCR (n-PCR) or quantitative real-time PCR(qPCR) of repetitive DNA sequences to achieve high sensitivity, PCR of a small, repetitive DNA sequence is preferred, because the efficiency of amplifying a small DNA fragment is higher than that of a large one. In addition, there are more template copies in a repetitive sequence per organism. Three repetitive DNA sequences are often used for the detection of T. gondii in biological samples, including the 35-copy B1 gene, the 300-copy 529 bp repeat element and the 110-copy internal transcribed spacer (ITS-1) or 18S rDNA gene sequences. A molecular detection method (conventional PCR) for T. gondii, targeting the B1 gene, wasfirst developed by Burg et al. (1989). This method has since been modified and adapted in different laboratories (Khalifa et al. 1994; Liesenfeld et al.1994; Bretagne et al. 1995; Pelloux et al. 1998; Contini et al. 2002, 2005; Reischl et al. 2003; Switaj et al. 2005; Bastien et al. 2007). The 529 bp repeat element was identified by Homan et al. (2000), and it was reported to be 10- to 100-times more sensitive than the B1 gene (Homan et al. 2000; Reischl et al.2003; Calderaro et al.

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2006). The 110-copy ITS-1 or18S rDNA has been used as the target in a few studies (Hurtado et al. 2001; Jauregui et al. 2001; Calderaro et al. 2006) and showed similar sensitivity to the B1 gene. To achieve higher sensitivity, n-PCR of B1 gene and ITS-1 sequences has been applied in some studies (Pelloux et al. 1998; Hurtado et al.2001; Jauregui et al. 2001; Contini et al. 2002, 2005; Bastien et al. 2007), both markers showed high sensitivity, with the detection level being as low as 1parasite (Hurtado et al. 2001; Jauregui et al. 2001; Calderaro et al. 2006). For a given repetitive sequence, n-PCR is more sensitive than conventional PCR. A case in point is that the n-PCR of the B1gene is more sensitive than the conventional PCR for the detection of T. gondii in amniotic fluid samples from congenital toxoplasmosis of human (Okay et al.2009). Recently, we employed a set of primers forn-PCR of T. gondii 18S rDNA, and found that it was more sensitive than the B1 gene (data not shown). The 18S rDNA marker is of particular interest, as it can distinguish several protozoan parasites which are closely related to T. gondii.

## DIAGNOSIS

The conventional diagnosis of T. gondii infection usually employs serological tests, bioassays in cats and/or mice, or a combination of the 2 approaches (Dubey and Beattie, 1988). In the past 2 decades, the diagnosis of T. gondii infection by direct detection of parasite-specific DNA in biological samples using PCR-based molecular methods has gained popularity. The molecular diagnosis is more sensitive and cost-effective than the conventional methods(Schoondermark-Van De Ven et al. 1994; Bessie`reset al. 2009).For the correct diagnosis of gestational toxoplasmosis, tests for antiToxoplasma gondii IgM and IgG and also IgG avidity (low or high) avidity test should be performed Examination of the placenta can often be the only indication that there has been congenital infection, in cases of late infection, lack of prenatal diagnosis or when antibodies are not detected in the neonate(Robert-Gangneux et al., 2010; Robert Gangneux and Darde 2012.Among the assays currently used for the biologic diagnosis of Toxoplasma gondii infection in neonates, specific IgM analysis of cord blood and neonatal serum had poor sensitivity(Robert-Gangneux et al., 2010).Furthermore, the detection of T. gondii DNA can also be verified in umbilical cord blood. Serological diagnosis represents the most widely used approach to define the stage of infection, whether current, recent, or past. However, despite its high sensitivity, these tests can provide ambiguous results. In such cases, direct detection of the parasite is necessary for a definitive diagnosis, which can be achieved classically by intraperitoneal inoculation of laboratory animals and inoculation of cells in culture. (Derouin, et.al 1987) The methods, however, are time consuming and expensive. Where serological assays are unreliable or when the clinical diagnosis is doubted, PCR-based techniques can be performed. (Bastien, et.al 2002) Detection of T gondii DNA using PCR minimizes the problems faced when using serology-based or cultured-based assays. It saves time and labor, offering the advantages of high sensitivity and specificity. PCR has been used to demonstrate the presence of Toxoplasma in various clinical samples: brain, whole blood, (Johnson et.al 1993) amniotic fluid, CSF, aqueous humor, and lymph nodes. (Johnson, et.al 1993) PCR is of utmost importance in diagnosing Toxoplasma infection in cases of immunosuppressive therapy or in patients with AIDS.O'(Driscoll, et.al 1991)Several approaches based on PCR have been developed and offer a significant improvement in diagnosis, especially for congenital toxoplasmosis. The current Toxoplasma PCR assays essentially targets two main loci. The

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first is the 35-repeat B1 gene.22 Several groups[i] have designed different sets of primers to different loca [ii] tons on the gene.((Jenum, et.al 1998,Pujol-Rique1999,James,et.al 1996) Another widely used target is the single-copy gene (P30), which codes for the major surface antigen P30.(Driscoll,et.al 1991) Here again, different sets of primers have been designed. (James,et.al 1996) As introduction of molecular diagnostic techniques is expected to improve the toxoplasmosis diagnosis serological diagnosis by enzyme-linked immune -sorbent assay (ELISA) method (Ichikawa-Seki, et.al 2015). In addition to the molecular diagnosis is an essential, accurate tool for the diagnosis of congenital toxoplasmosis and to evaluate the prevalence of Toxoplasma reactivation when the detection of circulating DNA is the only clue to its reactivation and its effect on gene expression in immune cytokines (Mousa, et.al 2021)

## CLINICAL SYMPTOME

Although the course of the primary infection is usually subclinical and the vast majority of infected human populations remain asymptomatic, the infection can cause significant morbidity and mortality in certain groups. This includes encephalitis, chorioretinitis, congenital infection and neonatal mortality ( Shahine L., Lathi R. 2015).Transmission to the fetus occurs in women who acquire their primary infection during gestation and can result in visual and hearing loss, mental and psychomotor retardation, seizures, hematological abnormalities, hepatosplenomegaly, or death (Basavaraju A. 2016) The global annual incidence of congenital toxoplasmosis was estimated to be 190,100 cases(Baqer N.N., Saheb E.J., Ahmed N.S. 2021).High burdens of congenital toxoplasmosis, which where estimated as the highest among all food-borne pathogens ( McLeod R., Cohen W., Dovgin S., Finkelstein L.,Boyer K.M. 2020).were seen in South America and in some Middle Eastern and low-income countries(Basavaraju A. 2016).Moreover, toxoplasma encephalitis due to reactivation of latent tissue cysts is the most common clinical presentation of toxoplasmosis among persons with AIDS Pomares C.,( Montoya J.G. 2016)(McLeod R., Cohen W., Dovgin S., Finkelstein L.,Boyer K.M. 2020) (Mesquita R.T., Ziegler A.P., Hiramoto R.M., VidalJ.E., Pereira-Chioccola V.L. 2010). The infection is typically observed in the later stages of human immunodeficiency virus (HIV)infection, when persons become severely immunosuppressed (Boshapor S.O., Kassem H.H. 2015) (Abdulkhaliq R.J., Mohammed S.T., WahhabAlkhateeb H.M., Abbas A.A. 2019).The incidence of encephalitis in AID Patients in the general population is directly related to the prevalence of anti-T. gondii antibodies(McLeod R., Cohen W., Dovgin S., Finkelstein L.,Boyer K.M. 2020)

## TREATMENT

The main accepted treatment for toxoplasmosis is a combination of sulfonamides and pyrimethamine (Petersen, 2007). Therapy with an association between sulfadiazine, pyrimethamine and folic acid is indicated for women with more than 18 weeks of pregnancy, for whom itis suspected or confirmed that they acquired acute infection at or after the 18th week of pregnancy, or a positive amniotic fluid PCR test result is documented, or an abnormal fetal ultra-sonography is suggestive of congenital toxoplasmosis (Maldonado et al., 2017).During the first trimester of pregnancy, in case of fetal infection, treatment with spiramycin is indicated (Kaye, 2011). The use of pyrimethamine is not recommended during the first trimester of pregnancy for having a teratogenic effect (Montoya and Remington,

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2008; Kaye,2011). In addition, pyrimethamine can suppress activity of the bone marrow. For these reasons, it is recommended to combine with folic acid (Montoya and Remington, 2008). Sulfadiazine and pyrimethamine act synergistically in blocking olate synthesis pathway by inhibiting the enzymes dihydropterate synthase and dihydrofolate reductase, which are essential for survival and parasite replication (Anderson, 2005) considered the analysis of the placenta an important tool to confirm the diagnosis of infection in newborns and thus provide early treatment.

**PREVENTION**

Avoid driving untreated water, Wear gloves when gardening and during any contact with soil or sand because it might be contaminated with cat feces that contain Toxoplasma. Wash hands with soap and water after gardening or contact with soil or sand. Teach children the importance of washing hands to prevent infection. Keep outdoor sandboxes covered. Feed cats only canned or dried commercial food or well-cooked table food, not raw or undercooked meats. Ensure that the cat litter box is changed daily. The Toxoplasma parasite does not become infectious until 1 to 5 days after it is shed in a cat's feces. If you are pregnant or immune compromised. Avoid changing cat litter if possible. If no one else can perform the task, wear disposable gloves and wash your hands with soap and water afterwards. Keep cats indoors to prevent them from hunting and reduce the chances they will become infected with Toxoplasma. Do not adopt or handle stray cats, especially kittens. Do not get a new cat while you are pregnant or immune compromised.

**CONCLUSION**

The application of an integrated approach, combining molecular detection and high resolution genetic characterization, should assist in enhancing our understanding of the molecular epidemiology, population genetics and phylogeny of T. gondii, which will be beneficial to the control of T. gondii transmission and a reduction of toxoplasmosis in humans and animals. And optimized semi-nested PCR of SAG2 gene, which is a reliable diagnostic technique with adequate sensitivity and specificity when used to detect T. gondii DNA in different clinical settings. The developed PCR method was able to detect as little as 12 ng/µL of T. gondii DNA and was useful to diagnose the disease in women who have had spontaneous abortions, HIV-positive patients, patients with leukemia and lymphoma, and infants with ocular infection. The analyzes detected Toxoplasma gondii DNA in 12.5% of the tested samples. Examination of the placenta can be an important tool in helping to diagnose congenital-infection early**.**

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