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## The prevention of toxoplasmosis gondii: A public health education for Prudentopolis, Brazil

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### Recommended Citation

Nevett, Stephen; Peterson, Jennifer; and Simonson, Amber, "The prevention of toxoplasmosis gondii: A public health education for Prudentopolis, Brazil" (1999). *College of Optometry*. 1285.  
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## The prevention of toxoplasmosis gondii: A public health education for Prudentopolis, Brazil

### Abstract

A educational brochure was designed to help educate the people in the region of Prudentopolis, Brazil about the possible means of preventing Toxoplasmosis gondii infection. The brochure resulted from the concern of several individuals after analyzing the data compiled from an Amigos Eye Care mission to Brazil. On this trip, it was noted that there was a significantly higher prevalence of ocular toxoplasmosis in the region as compared to the United States.

### Degree Type

Thesis

### Degree Name

Master of Science in Vision Science

### Committee Chair

Salisa Williams

### Subject Categories

Optometry

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**The Prevention of *Toxoplasmosis Gondii*:  
A Public Health Education for Prudentopolis, Brazil**

By

Stephen Nevett  
Jennifer Peterson  
Amber Simonson

A thesis submitted to the faculty of the  
College of Optometry  
Pacific University  
Forest Grove, Oregon  
for the degree of  
Doctor of Optometry  
May 22, 1999

Advisor:

Salisa Williams, O.D.

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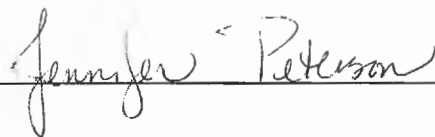
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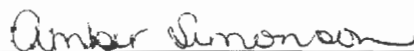
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Jennifer Peterson



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Amber Simonson



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Salisa Williams, O.D.

## The Prevention of *Toxoplasmosis Gondii*:

### A Public Health Education for Prudentopolis, Brazil

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#### Biographical Data

##### Stephen Nevett

Stephen Nevett is from Abbotsford, B.C. He received his Bachelor of Science Honors in Biology from Queen's University in Kingston, Ontario in 1994. Stephen attended Pacific University College of Optometry from 1995-1999, where he was a member of Amigos Eye Care, the National Optometric Student Association, and BSK. Stephen plans on practicing in the United States for a few years before returning to British Columbia.

##### Jennifer Peterson

Jennifer Peterson is from Blackfoot, ID. She attended Albertson College of Idaho in Caldwell, ID from 1991-1995 where she graduated with a Bachelor of Science in Zoology and a minor in music. She attended Pacific University College of Optometry from 1995-1999. While at Pacific University, Jennifer was an AFOS Liaison, a member of Amigos, and a member of BSK. Jennifer's future plans include a four year commitment in the military as an optometrist and a possibility of joining her father's optometric practice in Blackfoot, ID.

##### Amber Simonson

Amber Simonson is from McCall, ID. She attended Boise State University in Boise, ID from 1991-1995. She received her Bachelor's Degree in Visual Science from the College of Arts & Sciences, Pacific University in May 1997. She attended Pacific University College of Optometry from 1995-1999 where she received her Doctor of Optometry degree from the College of Optometry, Pacific University, Forest Grove, OR. While at Pacific University College of Optometry, Amber was a member of Amigos Eye Care, BSK, COVD, AOSA, and AOAPAC. Amber plans to work in a private practice in Boise, ID, specializing in contact lenses and sports vision.

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Abstract

A educational brochure was designed to help educate the people in the region of Prudentopolis, Brazil about the possible means of preventing *Toxoplasmosis gondii* infection. The brochure resulted from the concern of several individuals after analyzing the data compiled from an Amigos Eye Care mission to Brazil. On this trip, it was noted that there was a significantly higher prevalence of ocular toxoplasmosis in the region as compared to the United States.

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**Acknowledgments**

Salisa Williams, O.D.: Advisor

**Blu Gravier**: Illustrator

**Chad Cleverly**: Translator

**Team Amigos Brazil**

Thank you all very much for all your hard work  
and effort to help make our thesis complete.



# The Prevention of *Toxoplasmosis Gondii*

## A Public Health Education For Prudentopolis, Brazil

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### INTRODUCTION

On a recent trip to southern Brazil, Amigos, a non-profit organization providing vision care services via Pacific University College of Optometry, conducted a vision clinic for the individuals of Prudentopolis, Brazil. This team consisted of fifteen optometry students and two licensed optometrists who had a face-to-face experience with the devastation caused by ocular toxoplasmosis. Approximately 4.5% of the patients seen in the Amigo's clinic showed some evidence of either past or current ocular toxoplasmosis' complications. This was quite surprising considering that past studies indicate the incidence of ocular toxoplasmosis is significantly lower in other regions of the world (only 0.6% in the United States).<sup>1</sup> A study by Robert B. Nussenblatt, M.D., reported the incidence of ocular toxoplasmosis in southern Brazil to be 17.7% (thirty times higher than elsewhere in the world).<sup>2</sup> In addition, a study done in southern Brazil in 1990, by Peter D. Glasner M.D. and fellow colleagues, found a significant incidence (17.7%) of ocular toxoplasmosis and a predominance of acquired cases rather than congenital.<sup>1</sup>

Because of the significant number of patients showing signs of toxoplasmosis, public health awareness would be beneficial to help decrease the risk of infection. An educational brochure was designed for the people of Prudentopolis, Brazil (Appendix A). This brochure, written in Portuguese and English, illustrates several methods by which individuals could adapt lifestyle changes to help decrease the risk of acquiring or transmitting *toxoplasmosis gondii*. Through heightened awareness, it is hoped that the future incidence of infestation and occurrence of ocular toxoplasmosis will decrease.

The idea for a toxoplasmosis prevention program was conceived as "Team Brazil" began noticing the alarming number of patients with toxoplasmosis ocular infection. Although most cases were inactive, those patients with *Toxoplasmosis gondii* infection had significantly reduced visual acuities in the infected eye (Appendix C).

Sixty of the 1,328 (4.5%) patients seen were positive for toxoplasma retinitis; a number much higher than the average found in the rest of the world, estimated to be only 0.6%.<sup>1</sup> In addition, as an eye care team traveling in a foreign country, we experienced many living and social situations such as the increased diet of meat, very little hand washing, and raw meat exposed to the environment which promote the transmission of *Toxoplasmosis gondii*.

On behalf of Amigos Eye Care, it was decided to make an effort towards decreasing the incidence of acquired and congenital toxoplasmosis in the area of Prudentopolis. An educational program consisting of both posters and pamphlets will be created and distributed in the town of Prudentopolis. Our hope is to educate the citizens of Prudentopolis, and in turn, reduce the incidence of this sight robbing disease.

### **AMIGOS TRIP - TEAM BRAZIL**

In March of 1997, seventeen Amigos Eye Care members from Pacific University College of Optometry traveled to Prudentopolis, Brazil, to conduct a five-day vision care clinic.

Exactly 1,328 patients received vision exams. A thorough case history was taken on each patient with the aid of translators, and both near and far visual acuities were taken, using Snellen distance charts and Lighthouse picture charts respectively. The team used a Retinomax autorefractor by Nikon to best estimate the patient's needed prescription and performed comprehensive ophthalmoscopy. Any abnormal finding demanded examination by one of the two attending doctors who prescribed medications for conjunctivitis and other diseases, dispensed ocular lubricants, educated patients on disease, and referred patients for cataract surgery. Dilated fundus exams and intraocular pressures were performed at this stage of the exam. Finally, after the doctors and interns were satisfied everything possible had been done for the patient's ocular status, the individual proceeded to the dispensary to receive, if needed, a pair of eyeglasses, previously donated to

Amigos by optometrists and service organizations, such as Lion's Club and Kiwanis Club.

During our exam of 1,328 patients (920 being hyperopes, 275 myopes and 133 emmetropes), the Amigos team found sixty people with ocular toxoplasmosis infection; twenty five males and thirty five females (Appendix A). Thirty of the hyperopes, twenty-four of the myopes, and six emmetropes had ocular toxoplasmosis. This gives an overall prevalence of ocular toxoplasmosis of 4.5%, with 5.1% in males and 4.2% in females. In addition, emmetropes had a 4.5% chance of having signs of ocular toxoplasmosis, and hyperopes had a 3.3% chance of having ocular toxoplasmosis. Of interesting note is that myopes showed an 8.7% ocular toxoplasmosis infection rate.

### **TOXOPLASMOSIS: GENERAL OVERVIEW**

Toxoplasmosis is a disease of birds and mammals caused by the obligate intracellular protozoan known as *Toxoplasmosis gondii*. *T. gondii* affects approximately 500 million humans throughout the world.<sup>3</sup> Although it is found in a large portion of the world's human population, relatively few people have the active disease, toxoplasmosis. Toxoplasmosis is a disease process with both systemic and ocular complications. Toxoplasmosis is not common in the United States (estimated to be 0.6%),<sup>1</sup> yet has significant occurrence in countries where: 1) cat food is not processed, 2) there is little or no refrigeration of food, and 3) there is common practice of consumption of raw meat (particularly that which comes into contact with insects or cats).<sup>4</sup>

Toxoplasmosis can be subdivided into two types: 1) congenital (infant infected in utero) and 2) acquired (through such means as ingestion of raw eggs, meat, or unpasteurized milk). The disease, whether acquired or congenital, may be in latent or recurring forms (under conditions of reduced host defenses, such as AIDS). The infection may be acute or chronic. The individual who has the *T. gondii* infection, may be symptomatic or asymptomatic. The disease has a kaleidoscope of characteristics; the following are but a few of the systemic manifestations associated with the active disease: subclinical lymphadenopathy, malaise, maculapapular skin rash, hepatosplenomegaly, lymphocytosis, encephalitis, pneumonitis, and myocarditis.<sup>5</sup>

Of primary concern to optometrists is the devastating ocular presentations. The diagnosis of ocular toxoplasmosis is reliant on the presence of a characteristic retinal lesion in conjunction with the results of specified laboratory tests. The characteristic retinal lesion is typically a unilateral whitish yellow lesion (often with an associated adjacent area of inactive chorioretinal scar) with associated hazy vitreous ("headlights in the fog"). Other ocular manifestations may include vitreous precipitates and debris, optic disc edema, mild granulomatous iritis/uveitis, localized vasculitis, retinal artery or vein occlusion, chorioretinal scars, cystoid macular edema, visual field defects, and choroidal neovascular membrane.<sup>2</sup>

Ocular toxoplasmosis is not a well understood disease. However, because of its devastating ocular involvement, it should be a concern to optometrists as primary eye care physicians.

Toxoplasmosis is caused by an obligate intracellular parasite which is hosted primarily by the feline. However, it can infect and be hosted by almost any other mammal. Toxoplasmosis is present throughout the world, yet is more endemic to warm, humid climates.<sup>6</sup> Because the organism, *Toxoplasmosis gondii*, is excreted from the cat in a form which remains viable for many months in the open environment, many mammals are susceptible to contact the oocysts found in soil, dirt, etc.<sup>6</sup> It can also be transmitted via mother to fetus, via blood transfusion, via ingestion of undercooked meat, or via unpasteurized milk, lab accident and raw eggs.<sup>6</sup> Unfortunately, few studies have been conducted to enlighten the medical community concerning the reasons for the high prevalence of ocular toxoplasmosis in southern Brazil or on the means/methods possible to help prevent its infestation.

### TRANSMISSION OF TOXOPLASMOSIS GONDII

The transmission of *toxoplasmosis gondii* can either be congenital or acquired. Most of the cases (80%) of toxoplasmosis are congenital. While ocular involvement is common, systemic involvement is rare. Concerning congenital toxoplasmosis, the incidence of fetal infection is related to the balance between the host and the parasite. This balance includes the maturity of the fetus's immune system and the number and virulence of the parasites transmitted. Due to these factors, the age of the fetus at the time of infection

determines the occurrence of congenital toxoplasmosis. The optimal conditions for transmission are the initial parasitemia that occurs before development of cellular immunity in the mother and a well-developed placental blood flow.<sup>7</sup> This condition expresses itself when the primary infection occurs after week ten.<sup>7</sup> Although this conclusion has been made after numerous studies on animals as well as humans, congenital toxoplasmosis should be suspected in neonates of mothers who acquire the infection at any time during pregnancy.<sup>8</sup>

Signs of the disease in neonates may include seizures, feeding problems, respiratory disease, and diarrhea.<sup>8</sup> Extensive destruction may occur in the central nervous system causing damage in the cortex, mid brain, pons, medulla, and spinal cord. Obstruction of the foramina of Monro or the aqueduct of Sylvius can result in internal hydrocephalus. Maternal infection early in pregnancy is usually associated with fetal death or severe diseases at birth. Infection later in pregnancy typically results in infants damaged at birth.<sup>3</sup> Only 11% of maternal infections result in infants damaged at birth. It is stated that 60% of infants are not affected, but 29% have subclinical infections that manifest as neurologic or sensory defects as the infant develops. Other complications may include spontaneous abortion, microcephalus, chorioretinitis, multiple organ involvement, and encephalitis.<sup>3</sup>

The acquired form of toxoplasmosis is less frequent (20%) and tends to show more systemic involvement while ocular involvement is less prevalent. Acquired toxoplasmosis may be clinically silent in the normal host. In fact, 20-70% of American adults have immunoglobulin G antibodies to *Toxoplasmosis gondii*.<sup>9</sup> The infection occurs by the ingestion of the tissue cysts in uncooked meat or ingestion of sporulated oocysts by hands or food contaminated by cat feces can cause inoculation. The capsule surrounding ingested cysts is digested by gastric juices; this permits viable trophozoites to invade intestinal mucosa and to disseminate throughout the body by way of blood and the lymphatics. Organ cell invasion produces foci of necrosis surrounded by intense inflammatory reaction with mononuclear cell infiltration. The spleen, liver, brain, lung, myocardium, and eye are most frequently involved.<sup>3</sup> The development of cysts and tissue calcifications may impair organ functioning. An early antibody response destroys many parasites before they form tissue cysts and supports cyst formation by the

remainder. The infection is typically limited to its mild or subclinical form for the majority of infected persons.<sup>7</sup>

In order to decrease transmission in the population, certain precautions should be taken. It is said that 25% of lamb and pork have been shown to contain tissue cysts. Beef, unpasteurized goat milk, and eggs have also been said to contain the parasite.<sup>4</sup> Improper hygiene practices such as uncovered food accessible to cockroaches and flies, as well as exposure to the feline population increases the risks of contracting toxoplasmosis. It is also important to realize that *T. gondii* can be transmitted by needle stick accidents involving blood, although this is rarely seen.<sup>3</sup>

### LIFE CYCLE OF TOXOPLASMOSIS GONDII

There are many stages in the life cycle of the *Toxoplasmosis gondii* parasite including oocysts, sporozoites, bradyzoites (tissue cysts) and tachyzoites (trophozoites).<sup>9</sup>

Tachyzoites are the invasive form of toxoplasmosis, causing acute infection. This is an obligate intracellular organism<sup>10</sup> that invades every form of the mammalian cell, with the exception of non-nucleated erythrocytes. The tachyzoites, crescent shaped and 2-3um X 6-7um in size, are ingested by a host, and move into the mammalian cells through either a mechanical or chemical process. Once inside the cell, the tachyzoite resides in vacuoles and multiplies via endodyogeny. In the tachyzoite form, the organism is susceptible to heat, freezing, and gastric secretions.<sup>9</sup>

In order to survive in the human body over long periods of time, the tachyzoite transforms into a bradyzoite, the encysted form. In this form, the parasite is protected from immune response of the hosts. These cysts are responsible for latent infection, as they may rupture in the intestine wall, and multiply as tachyzoites within the lamina propria, resulting in active retinitis especially in an immunocompromised host.<sup>9</sup>

The oocyst, the sexual form of toxo, is 9-11um X 11-14um and contains four sporozoites.<sup>11</sup> The oocyst is produced only in the cat. The cat accumulates bradyzoites or oocysts from eating infected raw meat, wild birds and mice; it then invades the epithelial cells of the cat's intestine and undergoes an enteroepithelial asexual cycle of division, known as schizogony. A sexual phase results, and the release of millions of oocysts

occurs 3-21 days after ingestion. These oocysts undergo sporogony, forming four sporozoites, at which point they are considered infective. The oocytes are released into the environment via feces, and are viable for up to two years. Humans may be infected directly from the cat feces, or by ingesting the intermediate hosts of pork, mutton, beef, chicken, or eggs. Once inside the human, digestive enzymes disrupt the oocyst wall, and toxoplasmosis organisms, in the form of tachyzoites are released. The tachyzoites again cause active inflammation via reactions with the immune system.<sup>9</sup>

## **DIAGNOSTIC TESTING FOR TOXOPLASMOSIS GONDII**

The diagnosis of toxoplasma retinitis is twofold, being based on both the presence of ocular lesion and confirmation by lab tests. *Toxoplasmosis gondii* must be differentially diagnosed with other causes of retinochoroiditis, such as sarcoidosis, tuberculosis, syphilis, and viral and fungal infections. In addition, if the toxo is believed to be congenital, differential diagnosis must be made with congenital herpes simplex virus, and cytomegalovirus.<sup>9</sup>

There are numerous tests for the presence of *Toxoplasmosis gondii* in the human body; most are serologic, however these can be difficult to interpret as approximately 20-70% of the United States population is a carrier of toxoplasmosis gondii with only a few expressing any symptoms.<sup>9</sup>

There are nine main tests for antibodies to toxoplasmosis in human blood:

### **A. Serologic Tests**

#### **1. Sabin-Fieldman Dye Test**

The standard for detection of *Toxoplasmosis gondii* antibodies, from which all other tests are evaluated. Live *Toxoplasma* is incubated with the sample serum. If anti-Toxoplasma antibody is present, no staining with methylene blue dye will occur, as the organisms cell membrane will be lysed and unable to hold the dye.<sup>9</sup>

#### **2. Indirect Fluorescent Antibody (IFA) Test**

The most widely used test, as it uses killed organisms, making it much safer for use in the human body. This test takes five days to show a positive result.<sup>9</sup>

### 3. Indirect Hemagglutination Test

This is a good screening test, but is not accurate for diagnosis, as it presents with many false-negatives. It is useful two to four weeks after acute infection, and involves red blood cells exposed to the antigen becoming sensitized to the appropriate antibody and agglutinating in the presence of that antibody.<sup>9</sup>

### 4. Complement Fixation

As a positive result does not necessarily indicate acute infection, nor a negative result ruling out past infection, this test is not recommended and is rarely used.<sup>9</sup>

### 5. ELISA (Enzyme-Linked Immunosorbant Assay) test

A very sensitive test that detects both IgG and IgM anti-toxoplasma antibodies in the blood. New versions utilize a double sandwich technique, eliminating many false positive results often caused by ANA and rheumatoid factor.<sup>9</sup>

### 6. Aqueous Human Antibody

This test is not used very often as it requires a paracentesis. It compares the ratio of anti-Toxoplasma antibodies in the aqueous humor with that in the serum. Specifically,

\_\_\_\_\_ [anti-toxo AB] \_\_\_\_\_ compared to [ in serum]  
[gamma globulin in aqueous]

note: [ ] denotes concentration

A result of one-half to two indicates no infection; the presence of ocular toxoplasmosis is indicated by a number of eight or higher.<sup>9</sup>

### 7. Toxoplasma Skin Test

Very similar to the Tuberculosis PPD Mantou test, 0.1ml antigen from a mouse or chick is injected into the skin and the results are read at 48-72 hours. If a induration of >10mm in diameter is found, the test is considered positive.<sup>9</sup>

## B. Non-serologic Tests

### 1. Mice inoculation

Mice are injected with blood samples from the specimen and the mouse tissue is examined four weeks later for evidence of *Toxoplasmosis gondii* cysts or trophozoites.<sup>3</sup>



## 2. Electron Microscope

Tissue sections/smears from the specimen are examined under the electron microscope for trophozoites during the acute stages of infection.<sup>3</sup>

### TREATMENT OF TOXOPLASMOSIS GONDII

Toxoplasmosis can be successfully treated. Success and opportunity varies with the individual (Appendix B). In immunocompetent people, toxoplasmosis is benign and self-limited and rarely requires treatment. In those who are immunocompromised in some way, toxoplasmosis can be life-threatening.

In particular, ocular toxoplasmosis treatment varies upon presentation. The decision to treat is based on the nature and location of the lesion on the retina. With infection, it is possible to get direct involvement of the fovea, papillomacular bundle, the optic nerve head, cystoid macular edema, and macular pucker.<sup>12</sup> Small peripheral lesions will usually heal spontaneously, while lesions in the posterior pole and large destructive lesions are treated regardless of location.<sup>9</sup> Large impending lesions should receive all four drug therapies which will be discussed subsequently.<sup>13</sup> Depending on the point of time in the life-cycle, certain drug therapies can be effective. Particular drugs that will be discussed shortly have shown to be effective against the tachyzoite in animal models, but there is no success if the parasite has reached the bradyzoite stage.<sup>7,9</sup>

Although there is no set standard or consensus of the best drug therapy at the time of this writing, there are several drug combinations that are being used.<sup>7</sup> Drug combinations may be synergistic, but at this time synergism has only been demonstrated for pyrimethamine and triple sulfa.<sup>13</sup> First, pyrimethamine and sulfadiazine both interfere with folic acid metabolism. These two drugs basically accomplish the same task, but at different levels in the metabolic pathway. Pyrimethamine is either administered as a single oral loading dose of 75 mg or two loading doses of 50 mg 12 hours apart followed by a treatment of a dosage of 25 mg twice daily.<sup>9</sup> Side effects of pyrimethamine are dose-related and reversible bone marrow suppression may occur in 12%. This suppression may result in anemia, leukopenia, or thrombocytopenia.<sup>13</sup> Gastrointestinal intolerance occurs in 50% of patients.<sup>9</sup>

Sulfadiazine is administered as a single oral 2 gm loading dose followed by a treatment at a dosage of 1 gm 4 times daily. Side effects include skin rashes, crystalluria, albuniuria, and hematuria.<sup>12</sup> If only one drug is chosen, it should be a trisulfapyrimidine because of its minimal expense, good tolerance, lack of spoilage, and simplicity of use.<sup>13</sup> Usual dosage is 1-1.5 gm given orally four times daily.<sup>13</sup> Clindamycin is also utilized alone or in a combination with pyramethamine and sulfadiazine. The dosage given is 300 mg orally 4 times daily.<sup>12</sup> It does not cross the blood-brain barrier. Side effects include pseudomembranous colitis, which is reported to occur in anywhere from 1/50,000 to 1/100,000 cases.<sup>9</sup> Patients should be instructed to discontinue use of the drug if they have four or more bowel movements per day than is normal for them.<sup>13</sup>

Corticosteroids are also used to minimize the inflammatory response, but again, some type of antibiotic must be used synergistically to avoid infection due to the depression of the immune system. Systemic steroids are contraindicated in AIDS patients, although sub-Tenon's injections have proven to be quite safe.<sup>12</sup>

Besides drug therapy, surgical treatment of toxoplasmosis is also available. Photocoagulation and cryotherapy may destroy *Toxoplasma* cysts as well as the tachyzoites. Photocoagulation does kill cysts of *T. gondii*, but it also damages normal retina and cannot be relied on to destroy all traces of *T. gondii*. Photocoagulation may be used when drug therapies have failed or if the patient cannot tolerate the medication.<sup>13</sup> The side effects of these particular treatments include retinal detachment, retinal hemorrhage, and vitreal hemorrhage.<sup>9</sup> A subsequent vitrectomy may be required to remove vitreous opacities.<sup>12</sup> Vitrectomy is only indicated for patients with dense membranes and extremely reduced vision.<sup>13</sup>

## CONCLUSION

It is hoped that through the distribution of the pamphlets to the people of Prudentopolis, Brazil, a reduction in both the prevalence and incidence of ocular toxoplasmosis infection will occur. With only a few minor lifestyle changes, it is believed the numbers can be lowered.

Future studies may warrant further data collection in other parts of the world to determine if there is a direct correlation between cultural practices and hygiene to levels of ocular toxoplasmosis infection. In addition, further

studies may investigate the prevalence of hyperopes with ocular toxoplasmosis infection, compared to the number of myopes with the infection. Our results showed a much higher percentage of myopes having toxoplasmosis as opposed to hyperopes. It would be interesting to determine if the myopia is a result of ocular toxoplasmosis infection, or if myopes are predisposed to the ocular infection.

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Appendices

Appendix A

Toxoplasmosis gondii Pamphlet

## What is Toxoplasmosis?



*What a healthy eye sees*



*What your eye might see if you are infected with Toxoplasmosis gondii*



*Inside of your eye if you have an ocular Toxoplasmosis gondii infection*

## How To Prevent Toxoplasmosis Gondii Infection



*Drink pasteurized milk*



*Wash your hands well, especially after dealing with uncooked meat or soil*



*Cook your meat and eggs thoroughly*



*Wash fruits and vegetables*



*Toxoplasmosis can be passed onto your unborn child! Expectant mothers should stay away from litter boxes, cats, rodents, and birds*



*Wear gloves when working in the soil*



*Boil unclean drinking water*



*Keep animals out of children's play areas*

## Appendix B

### VARYING TREATMENTS OF *TOXOPLASMOSIS GONDII*<sup>7,12</sup>

Type of Infection	Medication	Dosage	Duration of Therapy
Pregnant women with acute toxoplasmosis (first 21 weeks of gestation or until term if fetus not infected)	Spiramycin	1 g every 8 hours without food	Until fetal infection documented or excluded at 21 weeks; if documented, in alternate months with pyrimethamine, leucovorin, and sulfadiazine
Confirmed fetal infection	<ol style="list-style-type: none"> <li>1. Pyrimethamine</li> <li>2. Sulfadiazine</li> <li>3. Leucovorin (folinic acid)</li> </ol>	<ol style="list-style-type: none"> <li>1. Loading dose: 100 mg per day in two divided doses for 2 days then 50 mg per day</li> <li>2. Loading dose: 75 mg/kg per day in two divided doses for 2 days, then 100 mg/kg per day in two divided doses (maximum 4 g per day)</li> <li>3. 10-20 mg daily</li> </ol>	<p>Same as above</p> <p>During and for 1 week after pyrimethamine</p>
Congenital infection in the infant	<ol style="list-style-type: none"> <li>1. Pyrimethamine</li> <li>2. Sulfadiazine</li> <li>3. Leucovorin</li> <li>4. Corticosteroids</li> </ol>	<ol style="list-style-type: none"> <li>1. Loading dose: 2 mg/kg per day for 2 days, then 1 mg/kg per day for 2 or 6 months, then this dose every Monday, Wednesday, Friday</li> <li>2. 100 mg/kg per day in two divided doses</li> <li>3. 10 mg three times weekly</li> <li>4. 1 mg/kg per day in two divided doses</li> </ol>	<ol style="list-style-type: none"> <li>1. 1 year</li> <li>2. 1 year</li> <li>3. During and for 1 week after pyrimethamine</li> <li>4. Until resolution of elevated CSF protein level or active chorioretinitis that threatens vision</li> </ol>



Active chorioretinitis in older children	<ol style="list-style-type: none"> <li>1. Pyrimethamine</li> <li>2. Sulfadiazine</li> <li>3. Leucovorin</li> <li>4. Corticosteroids</li> </ol>	<ol style="list-style-type: none"> <li>1. Loading dose: 2 mg/kg per day (maximum 50 mg) for 2 days, then maintenance, 1 mg/kg per day (maximum 25 mg)</li> <li>2. Loading dose: 75 mg/kg, then maintenance, 50 mg/kg every 12 hours</li> <li>3. 10-20 mg three times weekly</li> <li>4. 1 mg/kg per day in two divided doses</li> </ol>	<ol style="list-style-type: none"> <li>1. Usually 1-2 weeks beyond the time that signs and symptoms have resolved</li> <li>2. Usually 1-2 weeks beyond the time that signs and symptoms have resolved</li> <li>3. During and for 1 week after pyrimethamine therapy</li> <li>4. Until resolution of elevated CSF fluid protein level or active chorioretinitis that threatens vision</li> </ol>
Ocular toxoplasmosis	<ol style="list-style-type: none"> <li>1. Pyrimethamine</li> <li>2. Sulfadiazine</li> <li>3. Folinic Acid</li> <li>4. Prednisone</li> </ol>	<ol style="list-style-type: none"> <li>1. Two 50 mg loading doses 12 hours apart, then 25 mg by mouth twice daily</li> <li>2. 2 gm loading dose, then 1 gm by mouth four times daily</li> <li>3. 3-5 mg by mouth twice weekly</li> <li>4. 20-40 mg by mouth once daily</li> </ol>	3-4 weeks

## Appendix C

### General Numbers Tested

Total Hyperopes = 920

Total Myopes = 275

Total Emmetropes = 133 (all based on OD refraction)

Total tested = 1328

Total Males tested = 484

Total Females tested = 841 (note: total males and females only equals 1325 as three sexes were not recorded. For stats purposes, 1 will be added to 484 and 2 to 841 to result in the 1328 patients. This is based on the 2:1 female to male patient ratio).

### Prevalence of Toxoplasmosis in Prudentopolis Brazil

Toxo in OD = 42

42 cases/1328 = 3.6% (36 per 1000)

Toxo in OS = 28

28 cases/1328 = 2.1% (21 per 1000)

Toxo in OU = 10

10 cases/1328 = 0.75% (7.5 per 1000)

Total prevalence [(OD+OS)-OU]/1328 = 4.5% (45 per 1000)

Males with toxo = 25

25/485 = 5.1% (51 per 1000 males)

Females with toxo = 35

35/843 = 4.2% (42 per 1000 females)

# hyperopes with ocular toxo (based on OD refraction) = 30

30/920 = 3.3% (33 per 1000 hyperopes)

30/1328 = 2.3% (23 per 1000 in general population)

# myopes with ocular toxo (based on OD refraction) = 24

24/275 = 8.7% (87 per 1000 myopes)

24/1328 = 1.8% (18 per 1000 in general population)

# emmetropes with ocular toxo (based on OD refraction) = 6

6/133 = 4.5% (45 per 1000 emmetropes)

6/1328 = 0.45% (4.5 per 1000 in general population)

## Appendix C continued

### StatView Summary of Prudentopolis Data

	age	sex	va od	va os	toxo.od	toxo.os	rx.od	rx.os
Mean	42.891	1.635	125.28	124.93	.032	.021	.756	.81
Std. Deviation	17.607	0.482	387.17	380.39	.175	.144	1.546	1.70
Minimum	4	1	0.00	0.00	0	0	-8.750	-16.25
Maximum	87	2	2000	2000	1	1	11.250	14.75
Range	83.000	1	2000	2000	1.000	1.000	20.000	31.00
Count	1319	1327	1328	1328	1328	1328	1304	1317
Sum	56573	2169	166376	165906	42	28	98	1061

where: Sex 1=male 2=female  
VA 20/# (snellen)  
Toxo 1=present 0=none