International Advanced Research Journal in Science, Engineering and Technology

6th National Conference on Science, Technology and Communication Skills – NCSTCS 2K23

Narula Institute of Technology, Agarpara, Kolkata, India

Vol. 10, Special Issue 3, September 2023

Dynamical behaviour of Giardiasis

Nikhilesh Sil¹, Baishali Chatterjee², Ranabir Paul³ and Dibyendu Biswas⁴

Faculty, Department of Basic Science and Humanities, Narula Institute of Technology, Kolkata, India¹

Student, Department of Electronics and Communication Engg., Narula Institute of Technology, Kolkata India^{2,3}

Faculty, Department of Mathematics, City College of Commerce and Business Administration, Kolkata, India⁴

Abstract: In this paper, we presented a modelling approach to investigate the dynamics of Giardiasis in humans and domestic animals coupled with a contaminated environment. We computed the basic reproduction number R0 and employed it in analysing the effect of initial transmission and the stability of disease when an outbreak occurs. Results show that even when $\eta = 0$, R0 = R0h is greater than 4, showing that person-to-person transmission is the most significant in the dynamics of Giardiasis. An increase in η increases the value of R0 to some extent. Numerical simulations show that whenever there is an outbreak of Giardiasis in humans and domestic animals, the disease is likely to persist in the first two months and thereafter it will start to slow down to disease-free-equilibrium.

Keywords: Giardiasis, Disease free equilibrium, Stability Analysis, Control.

I. INTRODUCTION

Giardiasis, caused by the protozoa Giardia duodenalis (also known as Giardia lamblia and Giardia intestinalis), is an enteric infection prevalent in low-resource settings. It typically manifests with symptoms like flatulence and watery diarrhea[1–4]. In the United States, this disease is most commonly observed among international travelers, wilderness enthusiasts, and daycare workers. While many cases are asymptomatic, some patients experience severe symptoms leading to dehydration and weight loss. Fortunately, treatment with nitroimidazole or anthelminthic medication is often highly effective. Giardia duodenalis is responsible for this infection, and it is transmitted through the excretion of cysts by infected animals into freshwater sources. These cysts remain infectious for weeks to months. Seven genetic assemblages of Giardia have been identified, with genotypes A and B known to infect humans. Human infection occurs through the ingestion of cysts via contaminated water or direct person-to-person contact. Remarkably, even a small number of cysts, as few as 10, can lead to an infection [5–7].

After being excreted in feces, Giardia cysts become immediately infectious to new hosts, requiring no maturation or latent period [8]. These cysts can survive in various environments, particularly in water and at lower temperatures [9–11]. Sexual transmission of giardiasis, particularly among men who have sex with men, is a well-documented form of oral-anal and fecal-oral transmission [12]. Inadequate hygiene and sanitation practices significantly contribute to transmission. In modern times, daycares have become hotspots for giardiasis infections, primarily due to inadequate handwashing practices when handling and changing diapers. Within the intestinal system, the cysts undergo excystation and release trophozoites. These trophozoites are pear-shaped flagellated protozoa with two nuclei. Giardiasis is the most prevalent enteric protozoal infection globally, affecting approximately 2% of adults and 8% of children in developed countries. Estimates suggest that nearly 33% of the population in developing countries has been infected with giardiasis. In the United States, it is estimated that roughly 1.2 million individuals are affected, with most cases going undetected due to carriers being asymptomatic.

Giardia lamblia, initially observed by Antonie van Leeuwenhoek in 1681 within his own stool sample [13], was once viewed with uncertainty regarding its pathogenicity. However, it is now recognized as the predominant cause of protozoan-induced diarrhea in both humans and a wide range of domestic and wild animals globally [14]. Infections can lead to a spectrum of clinical manifestations, varying from asymptomatic colonization to acute or chronic diarrhea [15]. Notably, symptomatic infections have been documented in millions across Asia, Africa, and Latin America [16,17].

In 2012, the Centers for Disease Control and Prevention (CDC) documented 15,223 cases [18]. The most impacted demographic was children aged 0 to 4, and the majority of cases were reported in the northwest United States. Peak incidence typically coincides with late summer and early fall, primarily driven by outdoor water activities.





International Advanced Research Journal in Science, Engineering and Technology

6th National Conference on Science, Technology and Communication Skills – NCSTCS 2K23

Narula Institute of Technology, Agarpara, Kolkata, India

Vol. 10, Special Issue 3, September 2023



Fig. 1 schematic illustration of the virus.[19]

Globally, giardiasis incidence has been estimated at 2.8×10^{-8} cases annually [20]. However, numerous epidemiological studies suggest that these rates may be significantly underestimated. Prevalence rates of giardiasis range from 10 to 50% in developing countries [21, 22] and from 2 to 5% in developed nations [23, 24]. This discrepancy may be attributed to the substantial number of asymptomatic carriers who, despite lacking symptoms, play a pivotal role in disease transmission. Notably, Giardia infection in both animals and humans has been linked to growth retardation [25-26].

II. CAUSE AND SYMPTOMS

Giardia lamblia, commonly found in the feces of both animals and humans, thrives in various environments, including contaminated food, water, and soil. These hardy parasites can endure outside a host for extended periods, and accidental ingestion of these organisms can lead to infection. The primary mode of giardiasis transmission is through the consumption of water containing G. lamblia. Contaminated water sources include swimming pools, spas, and natural bodies of water like lakes. Such contamination often results from sources such as animal feces, diapers, and agricultural runoff. While acquiring giardiasis through food is less common due to heat effectively killing the parasites, poor hygiene practices during food handling or consumption of produce rinsed with contaminated water can facilitate the spread of the parasite. Additionally, giardiasis can spread through personal contact, such as unprotected sexual intercourse, facilitating transmission from one person to another. Activities like changing a child's diaper or exposure to the parasite while working in a daycare center are common routes of infection, particularly since children are at a heightened risk due to their exposure to feces during diaper changes or potty training. Giardia assumes a protective spore-like form known as cysts, which can endure harsh environmental conditions. These cysts are released through the bowel movements of infected individuals or animals. Giardiasis is transmitted when people accidentally ingest the parasite or its cysts, and astonishingly, just one to ten cysts are sufficient to cause infection. To provide perspective, approximately one million cysts could fit beneath a fingernail [27].

A person can become infected by several ways [28]:

• Ingesting contaminated drinking or recreational water.

[•] Touching their mouth with contaminated hands.

[•] Putting something in their mouth that has come into contact with the droppings of infected animals or the stool of infected humans.

[•] Eating raw or undercooked food that is contaminated.

[•] Inadequately washing their hands before preparing food, before eating, and after toileting or diapering.

[•]Exposure to the feces of an infected individual through sexual contact.

International Advanced Research Journal in Science, Engineering and Technology

6th National Conference on Science, Technology and Communication Skills – NCSTCS 2K23

Narula Institute of Technology, Agarpara, Kolkata, India

Vol. 10, Special Issue 3, September 2023

III. SYMPTOMS OF GIARDIASIS

Symptoms of giardiasis typically manifest within seven to 10 days after exposure, although onset can occur as early as three days or as late as 25 days afterward. These symptoms usually endure for two to six weeks but may persist for an extended period in some cases. Prolonged Giardia infection can lead to complications, such as arthritis or damage to the intestinal lining [29]. It's important to note that some individuals infected with Giardia may remain asymptomatic but still carry and transmit the disease. If you exhibit signs and symptoms of illness and have potential exposure to sources of Giardia parasites, it is advisable to seek medical attention [30]. Certain individuals can harbor Giardia parasites without displaying any symptoms. Symptoms of giardiasis typically become apparent one to two weeks after exposure and commonly include [31]: • Fatigue • Nausea • Diarrhea or greasy stools • Vomiting • Bloating and abdominal cramps • Weight loss • Excessive gas • Headaches • Abdominal pain

IV.DEVELOPMENT OF MATHEMATICAL MODEL

The model considers two populations, namely: humans and domestic animals coupled with contaminated water and food in the environment. There is a natural death rate in each stage because the infection may take a long time, and therefore individual may die naturally. The mode of transmission of giardiasis is the environment to host, host to host, and host to the environment.

$$\frac{dS_{h}}{dt} = \lambda h - \beta S_{h}I_{h} - \beta S_{h}W - \mu_{h}S_{h}$$
$$\frac{dI_{h}}{dt} = \beta S_{h}I_{h} + \beta S_{h}W - \gamma I_{h} - \mu_{h}I_{h}$$
$$\frac{dW}{dt} = \alpha I_{h} - \mu W$$
$$\frac{dR_{h}}{dt} = \gamma I_{h} - \mu_{h}R_{h}$$



Fig. 2 Schematic illustration of the interaction between the virus and target cells. [32]





International Advanced Research Journal in Science, Engineering and Technology

6th National Conference on Science, Technology and Communication Skills – NCSTCS 2K23

Narula Institute of Technology, Agarpara, Kolkata, India

Vol. 10, Special Issue 3, September 2023

TABLE 1 PARAMETERS AND THEIR DESCRIPTION

Parameter	Description of the Parameters
S_h	Susceptible Human population
I_h	Infected Human population
β	Disease Transmitted from Infected Human
ρ	Susceptible Individual become infected with Contaminated Water
W	Infected water(Parasite)
μ_{h}	Natural Death Rate of Human
α	Infected Individuals Spread Pathogens into Water at a rate
μ	Natural decay rate of parasite caused Giardia from water

V. EXISTENCE AND LOCAL STABILITY ANALYSIS OF THE EQUILIBRIUM POINTS

There are five equilibrium points of the given system. The equilibrium points are given by

$$E_0(0,0,0,0), E_1(\frac{\lambda_h}{\mu_h},0,0,0), E_2(S_h^2, I_h^2, W^2, 0),$$

where,

$$S_h^2 = \frac{\mu(\gamma + \mu_h)}{(\rho \alpha + \mu \beta)}, \qquad I_h^2 = \frac{\rho S_h W}{(\gamma + \mu_h - \beta S_h)}, W = \frac{\alpha I_h}{\mu}$$

 $E_3(S_h^3, I_h^3, 0, R_h^3),$ where,

$$S_h^3 = \frac{(\gamma + \mu_h)}{\beta}, \qquad I_h^3 = \frac{\beta \lambda_h - \mu(\gamma + \mu_h)}{\beta(\gamma + \mu_h)}, R_h^3 = \frac{\gamma I_h}{\mu_h}$$

and the interior point equilibrium is $E^*(\mathbf{S}^*_h, \mathbf{I}^*_h, \mathbf{W}^*, \mathbf{R}^*_h)$ **Lemma 1**: $E_1 = (\frac{\lambda_h}{u_h}, 0, 0, 0)$; This equilibrium point is LAS if $\mu(\gamma + \mu_h + \mu) - \beta \lambda_h > 0$, and $\mu_h \gamma \mu + \mu^2_h \mu - \mu \beta \lambda_h - \rho \lambda_h \alpha > 0$.

Proof. The eigenvalues of the matrix corresponding to the equilibrium point $E_1 = \left(\frac{\lambda_h}{u_h}, 0, 0, 0\right)$ are $-\mu_h$, $-\mu_h$, and other two eigenvalues will have negative real part if

 $\mu(\gamma + \mu_h + \mu) - \beta \lambda_h > 0, and \ \mu_h \gamma \mu + \mu^2{}_h \mu - \mu \beta \lambda_h - \rho \lambda_h \alpha > 0$ Therefore the equilibrium $E_1 = \left(\frac{\lambda_h}{u_h}, 0, 0, 0\right)$ is always stable under the above condition.

Lemma 2: $E_2 = (S_h^2, I_{h'}^2 W^2, 0)$ This equilibrium point is stable.

Proof. The eigen values of the matrix corresponding to the equilibrium point is $(-\mu_h)$, and two other eigen values are given by the characteristic equation, $\lambda^3 + a1\lambda^2 + a2\lambda + a3 = 0$

$$a1 = \beta I_{h}^{2} + \rho W^{2} + 2\mu_{h} - \beta S_{h}^{2} + \gamma + \mu$$

$$a2 = -2\beta^{2}S^{2}{}_{h}I^{2}{}_{h} - S^{2}{}_{h}(\rho W^{2}\beta + \beta\mu_{h} + \beta\mu - \rho\alpha + \beta\rho W^{2}) + I^{2}{}_{h}(\beta\gamma + \beta\mu_{h} + \beta\mu)$$

$$+ (\rho W^{2}\gamma + \mu_{h}\gamma + \rho W^{2}\mu_{h} + \mu^{2}{}_{h} + \gamma\mu + 2\mu_{h}\mu + \rho W^{2}\mu)$$

International Advanced Research Journal in Science, Engineering and Technology

6th National Conference on Science, Technology and Communication Skills – NCSTCS 2K23

Narula Institute of Technology, Agarpara, Kolkata, India

Vol. 10, Special Issue 3, September 2023

 $a3 = (\mu\gamma\beta + \mu u_h\beta)I_h^2 - S_h^2(-\mu_h\rho\alpha + \mu\beta\mu_h) + (\gamma\mu\rho W^2 + \mu\rho\mu_h W^2 + \mu\mu_h^2 + \mu\gamma \mu_h)$ The equilibrium point *E*₂ is LAS if *a*1 > 0, *a*3 > 0 and *a*1*a*2 - *a*3 > 0

Lemma 3: For $E_3 = (S_h^3, I_h^3, 0, R_h^3)$ This equilibrium point is LAS stable if A>0, B>0 and AB-C>0.

Where $A = \beta I_h^3 + 2\mu_h - \beta S_h^3 + \gamma + \mu$ $B = -2\beta^2 S_h^3 I_h^3 - S_h^3 (\beta\mu_h + \beta\mu - \rho\alpha) + I_h^3 (\beta\gamma + \beta\mu_h + \beta\mu) + (\mu_h\gamma + \mu_h^2 + \gamma\mu + 2\mu_h\mu)$ $C = (\mu\gamma\beta + \mu\mu_h\beta)I_h^3 - S_h^3 (-\mu_h\alpha\rho + \mu\beta\mu_h) + (\mu_h^2 + \mu\gamma\mu_h)$

Proof. The eigenvalues of the matrix corresponding to the equilibrium point *is* $(-\mu_h)$ and other three eigenvalues are given by the characteristic equation, $\lambda^3 + A\lambda^2 + B\lambda + C = 0$, Where,

$$A = \beta I_h^3 + 2\mu_h - \beta S_h^3 + \gamma + \mu_h$$

 $B = -2\beta^{2}S^{3}{}_{h}I^{3}{}_{h} - S^{3}{}_{h}(\beta\mu_{h} + \beta\mu - \rho\alpha) + I^{3}{}_{h}(\beta\gamma + \beta\mu_{h} + \beta\mu) + (\mu_{h}\gamma + \mu^{2}{}_{h} + \gamma\mu + 2\mu_{h}\mu)$ $C = (\mu\gamma\beta + \mu\mu_{h}\beta)I_{h}^{3} - S_{h}^{3}(-\mu_{h}\rho\alpha + \mu\beta\mu_{h}) + (\mu\mu_{h}^{2} + \mu\mu_{h}\gamma)$

Therefore the equilibrium point E_3 is LAS if A>0, B>0 and AB-C>0.

Lemma 4: The interior point equilibrium is, $E^*(S_h^*, I_h^*, W^*, R_h^*)$, which is LAS if

Proof. The eigenvalues of the matrix corresponding to the equilibrium point $is(-\mu_h)$ and two other eigenvalues are given by the characteristic equation, $\lambda^3 + a1\lambda^2 + a2\lambda + a3=0$

$$a1 = \beta I_{h}^{*} + \rho W^{*} + 2\mu_{h} - \beta S_{h}^{*} + \gamma + \mu$$

$$a2 = -2\beta^{2}S^{*}{}_{h}I^{*}{}_{h} - S^{*}{}_{h}(\rho W^{*}\beta + \beta\mu_{h} + \beta\mu - \rho\alpha + \beta\rho W^{*}) + I^{*}{}_{h}(\beta\gamma + \beta\mu_{h} + \beta\mu)$$

$$+ (\rho W^{*}\gamma + \mu_{h}\gamma + \rho W^{*}\mu_{h} + \mu^{2}{}_{h} + \gamma\mu + 2\mu_{h}\mu + \rho W^{*}\mu)$$

 $a3 = (\mu\gamma\beta + \mu\mu_h\beta)I_h^* - S_h^*(-\mu_h\alpha\rho + \mu\beta\mu_h) + (\gamma\mu\rho W^* + \mu\rho\mu_h W^* + \mu\mu_h^2 + \mu\mu_h\gamma)$

VI. CONCLUSION

The last two years have been really challenging the COVID-19 pandemic changed so much about the way we lived and worked. Now, just as we're starting to ease back into regular life, giardiasis started its infection. The majority of presenting patients will be non-toxic and may only require oral rehydration for initial fluid resuscitation. In more severe cases, intravenous (IV) fluids may be needed [33].

In this research article, we used a modelling approach to investigate the dynamics of giardiasis coupled with a contaminated environment. To study the effect of initial transmission of the disease we find the equilibrium point of the given system of equations and find the existence condition of the equilibrium and then we analysed the stability of the several equilibrium points [30-34]. The analysis of the stability of equilibrium points indicates that both the disease-free equilibrium and endemic equilibrium of the model system are locally and globally asymptotically stable.

REFERENCES

- Adam, E. A., Yoder, J. S., Gould, L. H., Hlavsa, M. C., & Gargano, J. (2016). Giardiasis outbreaks in the United States, 1971–2011. Epidemiology & Infection, 144(13), 2790-2801.
- [2]. Minetti, C., Chalmers, R. M., Beeching, N. J., Probert, C., & Lamden, K. (2016). Giardiasis. Bmj, 355.
- [3]. Guzman-Herrador, B., Carlander, A., Ethelberg, S., Freiesleben de Blasio, B., Kuusi, M., Lund, V., ... & Nygård, K. (2015). Waterborne outbreaks in the Nordic countries, 1998 to 2012. Eurosurveillance, 20(24).
- [4]. Enserink, R., Scholts, R., Bruijning-Verhagen, P., Duizer, E., Vennema, H., de Boer, R., ... & van Pelt, W. (2014). High detection rates of enteropathogens in asymptomatic children attending day care. PloS one, 9(2), e89496.
- [5]. Coffey, C. M., Collier, S. A., Gleason, M. E., Yoder, J. S., Kirk, M. D., Richardson, A. M., ... & Benedict, K. M. (2021). Evolving epidemiology of reported giardiasis cases in the United States, 1995–2016. Clinical infectious diseases, 72(5), 764-770.



International Advanced Research Journal in Science, Engineering and Technology

6th National Conference on Science, Technology and Communication Skills – NCSTCS 2K23



Vol. 10, Special Issue 3, September 2023

- [6]. Hall, A. (1994). Giardia infections: Epidemiology and nutritional consequences. Giardia: from molecules to disease., 251-280.
- [7]. Rendtorff, E. C., & Holt, C. J. (1954). The experimental transmission of human intestinal protozoan parasites. IV. Attempts to transmit Endamoeba coli and Giardia lamblia cysts by water. American journal of hygiene, 60(3), 327-38.
- [8]. Thompson, R. A. (2008). Giardiasis: modern concepts in control and management. Annales Nestlé (English ed.), 66(1), 23-29.
- [9]. Grit, G. H., Bénéré, E., Ehsan, A., De Wilde, N., Claerebout, E., Vercruysse, J., ... & Geurden, T. (2012). Giardia duodenalis cyst survival in cattle slurry. Veterinary parasitology, 184(2-4), 330-334.
- [10]. Olson, M. E., Goh, J., Phillips, M., Guselle, N., & McAllister, T. A. (1999). Giardia cyst and Cryptosporidium oocyst survival in water, soil, and cattle feces (Vol. 28, No. 6, pp. 1991-1996). American Society of Agronomy, Crop Science Society of America, and Soil Science Society of America.
- [11]. DeRegnier, D. P., Cole, L., Schupp, D. G., & Erlandsen, S. L. (1989). Viability of Giardia cysts suspended in lake, river, and tap water. Applied and environmental microbiology, 55(5), 1223-1229.
- [12]. Escobedo, A. A., Almirall, P., Alfonso, M., Cimerman, S., & Chacín-Bonilla, L. (2014). Sexual transmission of giardiasis: a neglected route of spread?. Acta Tropica, 132, 106-111.
- [13]. Ford, B. J. (2005). The discovery of Giardia. Microscope-Chicago, 53(4), 161-167.
- [14]. Cacciò, S. M., & Sprong, H. (2011). Epidemiology of giardiasis in humans (pp. 17-28). Springer Vienna.
- [15]. Leung, A. K., Leung, A. A., Wong, A. H., Sergi, C. M., & Kam, J. K. (2019). Giardiasis: an overview. Recent patents on inflammation & allergy drug discovery, 13(2), 134-143.
- [16]. Torgerson, P. R., Devleesschauwer, B., Praet, N., Speybroeck, N., Willingham, A. L., Kasuga, F., ... & de Silva, N. (2015). World Health Organization estimates of the global and regional disease burden of 11 foodborne parasitic diseases, 2010: a data synthesis. PLoS medicine, 12(12), e1001920.
- [17]. Lane, S., & Lloyd, D. (2002). Current trends in research into the waterborne parasite Giardia. Critical reviews in microbiology, 28(2), 123-147.
- [18]. Savioli, L., Smith, H., & Thompson, A. (2006). Giardia and Cryptosporidium join the 'neglected diseases initiative'. Trends in parasitology, 22(5), 203-208.
- [19]. Adam, R. D. (2014). Protozoa: Giardia lamblia. In Hazards and Diseases (pp. 37-44). Elsevier.
- [20]. Daly, E. R., Roy, S. J., Blaney, D. D., Manning, J. S., Hill, V. R., Xiao, L., & Stull, J. W. (2010). Outbreak of giardiasis associated with a community drinking-water source. Epidemiology & Infection, 138(4), 491-500.
- [21]. Pires, S. M., Fischer-Walker, C. L., Lanata, C. F., Devleesschauwer, B., Hall, A. J., Kirk, M. D., ... & Angulo, F. J. (2015). Aetiology-specific estimates of the global and regional incidence and mortality of diarrhoeal diseases commonly transmitted through food. PloS one, 10(12), e0142927.
- [22]. Painter, J. E., Gargano, J. W., Collier, S. A., & Yoder, J. S. (2015). Giardiasis surveillance—United States, 2011–2012. Morbidity and Mortality Weekly Report: Surveillance Summaries, 64(3), 15-25.
- [23]. Yoder, J. S., Gargano, J. W., Wallace, R. M., & Beach, M. J. (2012). Giardiasis surveillance—United States, 2009–2010. Morbidity and Mortality Weekly Report: Surveillance Summaries, 61(5), 13-23.
- [24]. Garba, C. M. G., & Mbofung, C. M. F. (2010). Relationship between malnutrition and parasitic infection among school children in the Adamawa Region of Cameroon. Pakistan Journal of nutrition, 9(11), 1094-1099.
- [25]. Al-Mekhlafi, M. H., Azlin, M., Nor Aini, U., Shaik, A., Sa'iah, A., Fatmah, M. S., ... & Norhayati, M. (2005). Giardiasis as a predictor of childhood malnutrition in Orang Asli children in Malaysia. Transactions of the Royal Society of Tropical Medicine and Hygiene, 99(9), 686-691.
- [26]. Farthing, M. J. G. (1984). Giardiasis: pathogenesis of chronic diarrhea and impact on child growth and development. Chronic diarrhea in children, 6, 253.
- [27]. Vivancos, V., González-Alvarez, I., Bermejo, M., & Gonzalez-Alvarez, M. (2018). Giardiasis: characteristics, pathogenesis and new insights about treatment. Current Topics in Medicinal Chemistry, 18(15), 1287-1303.
- [28]. SM, C. (2008). Molecular epidemiology of giardiasis. Mol Biochem Parasitol, 160, 75-80.
- [29]. Kucik, C. J., Martin, G. L., & Sortor, B. V. (2004). Common intestinal parasites. American family physician, 69(5), 1161-1169.
- [30]. McCluskey, C. C. (2006). Lyapunov functions for tuberculosis models with fast and slow progression.
- [31]. Agarwal, A. K., Singh, M., Gupta, N., Saxena, R., Puri, A., Verma, A. K., ... & Saxena, K. C. (1994). Management of giardiasis by an immuno-modulatory herbal drug Pippali rasayana. Journal of ethnopharmacology, 44(3), 143-146.
- [32]. Ramirez, N. J., Posadas-Cantera, S., Caballero-Oteyza, A., Camacho-Ordonez, N., & Grimbacher, B. (2021). There is no gene for CVID—novel monogenetic causes for primary antibody deficiency. Current Opinion in Immunology, 72, 176-185.
- [33]. Korobeinikov, A. (2004). Lyapunov functions and global properties for SEIR and SEIS epidemic models. Mathematical medicine and biology: a journal of the IMA, 21(2), 75-83.
- [34]. Korobeinikov, A. (2007). Global properties of infectious disease models with nonlinear incidence. Bulletin of Mathematical Biology, 69, 1871-1886.

