

Purpose:

To enhance student proficiency in conducting library research of peer-reviewed primary research articles and to increase proficiency in formatting papers for submission to scientific journals. And, hopefully to learn some stuff about parasites.

Overview:

Students will select their parasite of choice and confirm this choice with the instructor by the date listed below. Students will use the library or internet resources to procure 3 primary research articles covering their chosen parasite. Write a short summary of each article. Write a literature cited page that includes your 3 journal articles in the style and format of the *Journal of Parasitology* published by the American Society of Parasitologists.

Instructions:

1. Choose a parasite by the end of class, Monday, 18 Sep 2017, tell Mr. Raymond the name of your parasite and annotate your choice of parasite on the sign-up sheet.
2. Use library or internet resources to obtain 3 primary research articles about your parasite. Note: articles must reference a volume number and page numbers. You may not use any articles published in any of the PLOS online journals: PLOS One, PLOS Biology, PLOS Medicine etc.
3. Save a copy of each article in pdf format (I don't want links to the document, the html format or any other format for that matter).
4. Use Times New Roman, 12 point font throughout.
 - a. Write a title page that includes the following information centered horizontally and vertically:
Your Name
BIOL 4473: Parasitology and Laboratory
University of the Incarnate Word
Date
 - b. Write a short summary of each article. Margins: left 1.5", right 1.0", top and bottom 1.0". The summary should include: 1. the title of the article and the names(s) of the author(s), 2. the organism or organisms being studied, 3. where the research was accomplished, 4. the primary experimental protocol used and 5. primary conclusions. (Examples of experimental protocols might be: PCR, ELISA, Cell Culture, Microscopic Examination etc. Basically, just briefly tell me what they did.) This can probably be accomplished in a few sentences. This summary should be written in your own words. I know that students in this class would not try to just copy the abstract word for word!
 - c. Write a literature cited page that includes proper citations for your 3 articles. (Use the style and format of the *Journal of Parasitology*.)
 - d. Convert the title page, summaries and literature cited page into a single (1) pdf document.
5. Send the pdf versions of your articles (3) and the pdf file containing the title page, summaries and literature cited page to me as email attachments. (So, your submission email should contain 4 pdf attachments). I will respond to your email when I receive it.

Due Date: I must receive your submission email by Monday, 06 Nov 2017, 12:00 P.M. (email stamp when received)

Grading Rubric:

	<u>Possible Points</u>	
Chose a parasite, confirm with Mr. Raymond, and annotate the sign-up sheet by deadline	0-5	5
Title page with required information, proper format	0-5	4
Articles are primary research articles	0-15	0
Articles are submitted as pdf files	0-15	15
Article summaries address required points and in proper format	0-30	24
Literature cited page in proper style and format	0-30	9

Total Possible Points **100** 57

Parasitology and Laboratory: Trypanosoma Brucei

Student's Name

BIOL 4473: Parasitology and Laboratory

University of the Incarnate Word

Monday October 6, 2017

Include your name on title page. -1

Title page, summaries and literature cited not submitted as a pdf document. -5

Articles are not primary research articles; they are review articles. -15

Genus and species names should be in italics. -1; Do not capitalize the species name.

Parasitology and Laboratory: Trypanosoma Brucei

Morrison, L.J. (2011). Parasite-driven Pathogenesis in Trypanosoma Brucei Infections. Parasite Immunology, 33, 448–455.

This article is titled "Parasite-motivated Pathogenesis in *Trypanosoma Brucei* Toxicities" and is written by L.J. Morrison. The report states that trypanosomes are of veterinary and medical importance because of the different diseases they cause in their mammalian hosts. For example, the two human-infective *Trypanosoma Brucei* which causes both chronic and acute illness. The author notes that thanks to recent research, we now comprehend the ways in which the initial reaction by the host influences the progression of the illness besides how disease severity is affected by the genetic variation of the condition. Nonetheless, the author notes that little attention is given in accepting how difference sickness outcome is affected by the genetic modification of *Trypanosoma brucei* in mammals.

This article is a **review** of the recent advances in the study of trypanosomes and how their genetic variation affects differential disease results. The report also presents an overview of the current literature regarding *Trypanosoma brucei* genes involvement in determining differential disease outcome and current approaches towards identifying its virulence genes. Lastly, the author discusses the possibility of using parasite genotype variation in examining the evolution perspective of virulence.

The author concludes that ways in which *Trypanosoma brucei* influences disease course are an essential research area regarding trypanosomes. Establishing how virulent strains of *trypanosoma brucei* cause clinical signs will enhance our knowledge of disease progression. According to the author, such a move will be necessary for answering some

of the lingering questions regarding trypanosome biology, boosting efforts of advancing ant- illness approaches. All available usable after a genomic process present a golden chance to start to understand disease processes. The author concludes that this area of study is advancing from a one-genetic factor investigation and is moving to focusing on identification of supervisory processes as well as genes. Advancement towards the identification of genes and regulatory processes has been made possible by modern technological advancements like multiple host gene and pathogen sequences, RNA sequencing, and in silico modeling among others. The author notes that there is a golden opportunity to include these strategies systematically to understand how the contact amid the host and the pest motivation ailment.

Ponte-Sucre, A. (2016). An Overview of Trypanosoma brucei Infections: An Intense Host-Parasite Interactions. Frontiers in Microbiology, 7, 2126.

This article by Alicia Ponte-Sucre provides an overview of disease caused by the pathogen, *trypanosoma brucei*. Specifically, the report provides an overview of *trypanosoma brucei rhodesiense* and *trypanosoma brucei gambiense*—the two primary causal means of the Human African Trypanosomiasis. These two etiologic agents are transmitted from the vector to the host by tsetse flies. Once in the vector, the parasite goes over multiple alterations which ready it to contaminate its anthropological host. Consequently, a transformational phase remains trypano-epimastigote and multiplication procyclic together with an infective, non- duplicative, and metacyclic system which grow within a salivary gland of tsetse flies. As an adaptive measure to survive in human hosts, metacyclic pathogens start to express and are covered by a thick layer of the Variant Surface Glycoprotein (VSG). Immediately after the parasite in metacyclic system enters

the body of the anthropological host, it starts to grow into the form of the bloodstream. Once it gets into the human body, the VSG activates a humoral protected reaction. To survive the humoral reaction and continue to develop in the host's bloodstream, the parasite mutates its outer cover occasionally by shedding the expressed VSG into the surroundings. This mutation helps the parasite evade the effects of the attack resulting from the activation of the immune system. Moreover, the parasite uses tools similar to quorum sensing in its transmission from the host to the vector.

This article is a detailed review of infections associated with *trypanosoma brucei*. The author analyzes the complex interaction between the parasite and the human host. This article is mainly a literature review in which the author examines previous research work regarding the topic. Throughout the report, the author cites several studies that were conducted by other people. More importantly, the author incorporates past and recent studies involving the contact amid ta host besides a pest in *Trypanosoma brucei* Contagions.

This author concludes that an antigenic difference remains to be a way through which trypanosomes deploy the anthropological habitats. Once *trypanosoma brucei* enters the human body, the immune system triggers a humoral response. However, the pathogen covers itself with VSG, which is the central antigenic molecule that covers the exterior of the plasma pathogens. The tools through which antigen swapping takes place are complicated, then possibly responsible for contagion chronicity. Consequently, the author concludes that renewal through VSG switching is an adaptation measure by *trypanosoma brucei* to evade attack by blood-borne antibodies. This adaptation enables the pathogen to survive in the bloodstream of the host and infect it. The author affirms that the VSG

switch affects the efficiency in the protective structure allowing the organism to survive within a hosts' body long enough to enable the diffusion towards a different host.

Although neither growing evolution to the spreading phases nor antigen switching is host determined, the host plays a role in the infection cycle. The host plays a role in the infection dynamics through genetic susceptibility, selection separate antigen types, besides a probable effect of host-dependent impacts on the pathogenicity of the parasite. The article concludes that the complex events involved indicate that trypanosomes employ a intensive plan which challenges the being of a well-established response from the human host.

Ralston, K. S., Kabututu, Z. P., Melehani, J. H., Oberholzer, M., & Hill, K. L. (2009). The Trypanosoma brucei Flagellum: Moving Parasites in New Directions. Annual Review of Microbiology, 63, 10.

Although there are many species of trypanosoma brucei, this article states that African trypanosomes are the most overwhelming animal as well as human pathogens. The two main trypanosome brucei sub-species; **T.b. gambiense** and **trypanosoma brucei Rhodesiense** causes the African sleeping sickness. If untreated early, the disease can be fatal and hundreds of infections occur every year. Tsetse flies swaps transmit trypanosoma brucei between insect and bloodstream forms. The pathogen is adapted to surviving in the bloodstream of the human host and also in the insect vector. There has been a lot of interest in the significance role of flagellum in the molting of parasites and connection to the vector's salivary gland epithelium. Recent research has discovered novel and conserved characteristics of the structure of flagellum of **T. brucei** and its composition.

This article is a review of past and current studies regarding the structure and composition of *trypanosoma brucei* flagellum. Although many studies have been conducted in the past regarding the importance of the flagellum for parasite motility and attachment to the vector's salivary gland epithelium, more research is required to unearth new drug targets. Throughout the article, the authors borrow extensively from previous studies and also from current research in analyzing the composition and structure of the flagellum of *T. brucei*. The first conclusion of the authors is that flagellar motility plays a vital role in disease pathogenesis and parasite development. Moreover, they state that *T. brucei* flagellum is an essential and multifunctional organelle that plays other necessary parts other than motility. Some of these roles include cell morphogenesis, immune evasion, host-parasite interaction, and cell division. The article also concludes that flagellar mutants have weak cell division in both the procyclic and bloodstream forms. Nonetheless, there are essential differences in phenotypes detected, revealing a flagellum functions that are specific to particular stages in the pathogens' lifecycle.

References

Morrison, L. J. (2011). Parasite-driven Pathogenesis in *Trypanosoma Brucei* Infections.

Parasite Immunology, 33, 448–455.

Insert a space between author's initials. -1

Do not put parentheses around publication year. -1

Capitalize only the first word and proper nouns within the title. -1

Genus and species names should be in italics. -1

Do not capitalize the species name. -1

Delete the comma following the journal name. -1

Delete the comma following the volume number and insert a colon. -1

Volume number and colon should be in bold print. -1

Ponte-Sucre, A. (2016). An Overview of *Trypanosoma brucei* Infections: An Intense

Host-Parasite Interaction. Frontiers in Microbiology, 7, 2126.

Do not put parentheses around the publication year. -1

Capitalize only the first word, the word following a colon, and proper nouns in title. -1

Genus and species names should be in italics. -1

Delete the comma following the name of the journal. -1

Delete comma following the volume number and insert a colon. -1

Ralston, K. S., Kabututu, Z. P., Melehani, J. H., Oberholzer, M., & Hill, K. L. (2009).

The *Trypanosoma brucei* Flagellum: Moving Parasites in New Directions. Annual

Review of Microbiology, 63, 10.

Except for the first author, the initials go in front of the last name. -1

Do not use the ampersand, write out the word "and". -1

Delete parentheses around publication year. -1

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Capitalize only the first word and proper nouns within the title. -1

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Include all page numbers. -1

Total: -21

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References

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