Trichomonas vaginalis

MEDICAL ACADEMY NAMED AFTER S.I. GEORGIEVSKY OF VERNADSKY CFU

DEPARTMENT OF MEDICAL BIOLOGY

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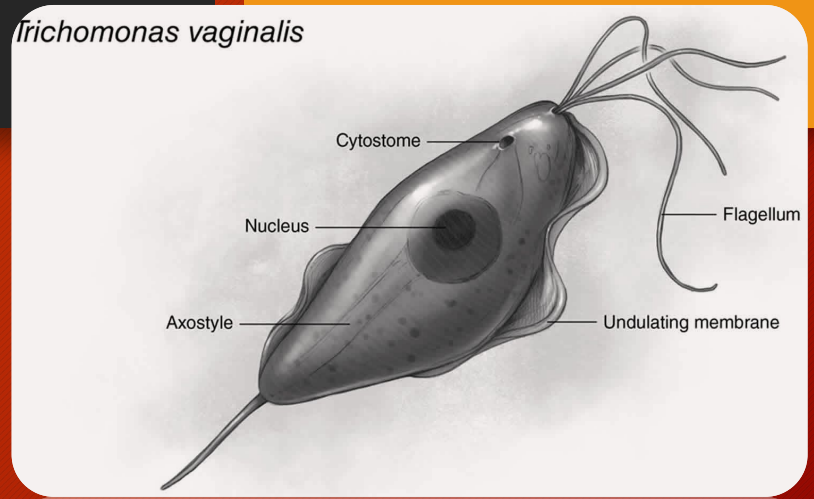
Trichomonas vaginalis

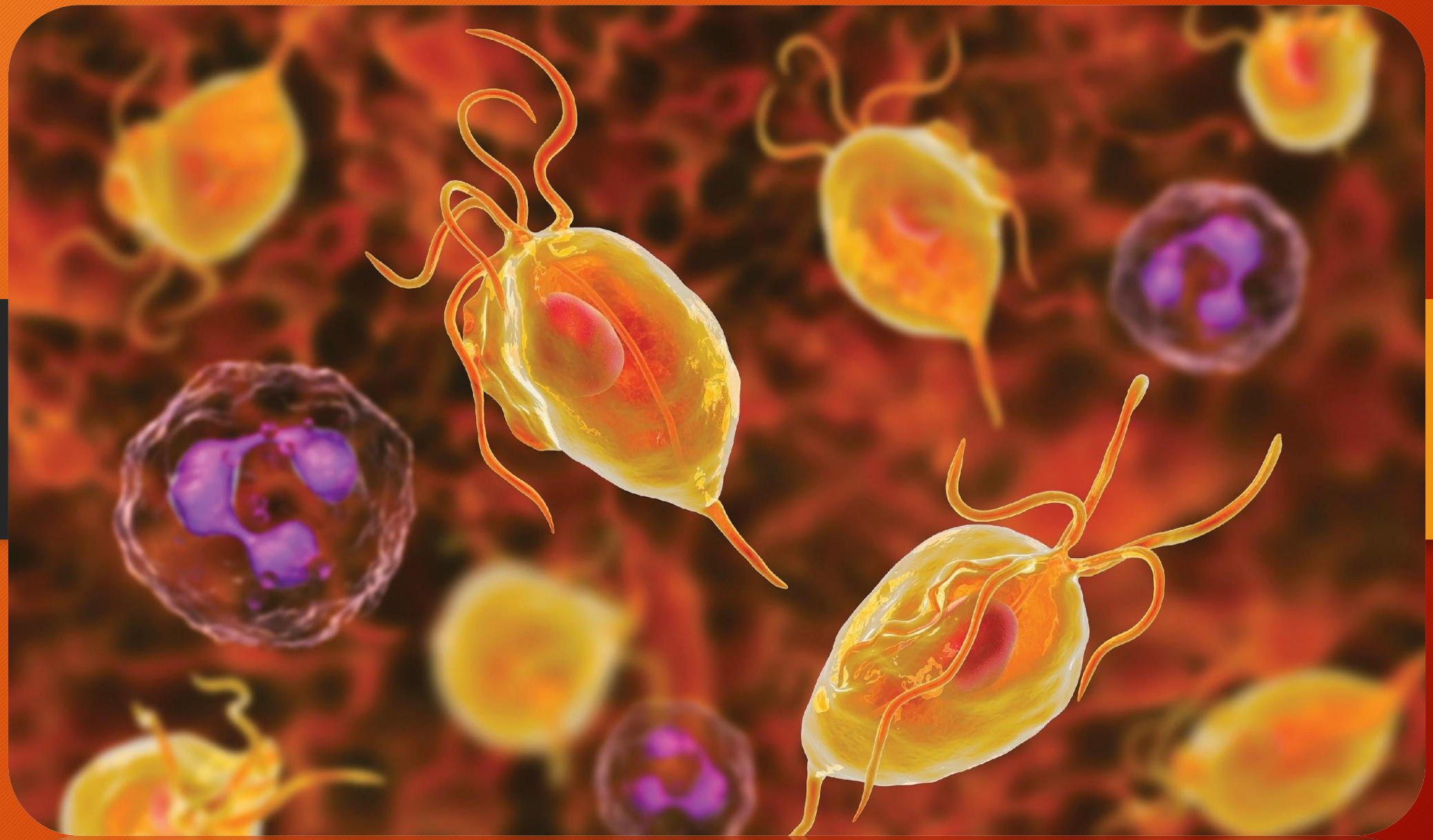
- Classification and Morphology;

Domain: Eukarya
Phylum: Metamonada
Class: Parabasalia
Order: Trichomonadida
Genus: *Trichomonas*
Species: ***T. vaginalis***
Binomial name ***Trichomonas vaginalis***

TRICHOMONAS VAGINALIS

IS AN ANAEROBIC, FLAGELLATED PROTOZOAN PARASITE AND THE CAUSATIVE AGENT OF TRICHOMONIASIS. IT IS THE MOST COMMON PATHOGENIC PROTOZOAN INFECTION OF HUMANS IN INDUSTRIALIZED COUNTRIES.[1] INFECTION RATES BETWEEN MEN AND WOMEN ARE SIMILAR WITH WOMEN USUALLY BEING SYMPTOMATIC, WHILE INFECTIONS IN MEN ARE USUALLY ASYMPTOMATIC. TRANSMISSION USUALLY OCCURS VIA DIRECT, SKIN-TO-SKIN CONTACT WITH AN INFECTED INDIVIDUAL, MOST OFTEN THROUGH VAGINAL INTERCOURSE. THE WHO HAS ESTIMATED THAT 160 MILLION CASES OF INFECTION ARE ACQUIRED ANNUALLY WORLDWIDE.[2] THE ESTIMATES FOR NORTH AMERICA ALONE ARE BETWEEN 5 AND 8 MILLION NEW INFECTIONS EACH YEAR, WITH AN ESTIMATED RATE OF ASYMPTOMATIC CASES AS HIGH AS 50%.[3] USUALLY TREATMENT CONSISTS OF METRONIDAZOLE AND TINIDAZOLE.





MORPHOLOGY

Unlike other parasitic protozoa (*Giardia lamblia*, *Entamoeba histolytica* etc.), *Trichomonas vaginalis* exists in only one morphological stage, a trophozoite, and cannot encyst. The *T. vaginalis* trophozoite is oval as well as flagellated, or "pear" shaped as seen on a wet-mount. It is slightly larger than a white blood cell, measuring 9 × 7 µM. Five flagella arise near the cytostome; four of these immediately extend outside the cell together, while the fifth flagellum wraps backwards along the surface of the organism. The functionality of the fifth flagellum is not known. In addition, a conspicuous barb-like axostyle projects opposite the four-flagella bundle. The axostyle may be used for attachment to surfaces and may also cause the tissue damage seen in trichomoniasis infections.

While *T. vaginalis* does not have a cyst form, organisms can survive for up to 24 hours in urine, semen, or even water samples.

•MECHANISM OF INFECTION

TRICHOMONAS VAGINALIS, A PARASITIC PROTOZOAN, IS THE ETIOLOGIC AGENT OF TRICHOMONIASIS, AND IS A SEXUALLY TRANSMITTED INFECTION.[2][6] MORE THAN 160 MILLION PEOPLE WORLDWIDE ARE ANNUALLY INFECTED BY THIS PROTOZOAN

HISTORY

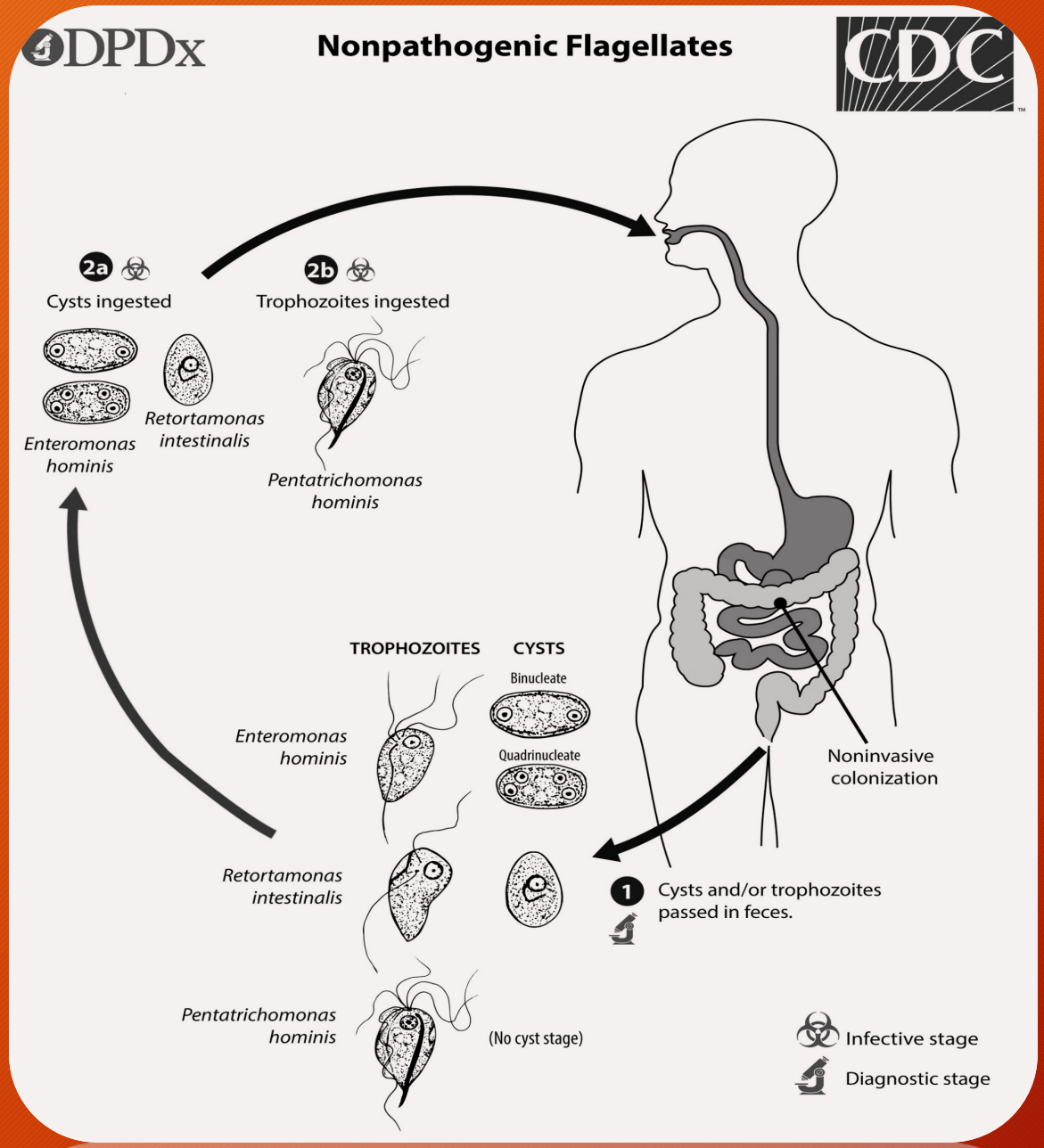
- Alfred Francois Donné (1801–1878) was the first to describe a procedure to diagnose trichomoniasis through "the microscopic observation of motile protozoa in vaginal or cervical secretions" in 1836. He published this in the article entitled, "Animalcules observés dans les matières purulentes et le produit des sécrétions des organes génitaux de l'homme et de la femme" in the journal, Comptes rendus de l'Académie des sciences.[5] As a result, the official binomial name of the parasite is Trichomonas vaginalis Donné.

COMPLICATIONS

- SOME OF THE COMPLICATIONS OF T. VAGINALIS IN WOMEN INCLUDE: PRETERM DELIVERY, LOW BIRTH WEIGHT, AND INCREASED MORTALITY AS WELL AS PREDISPOSING TO HIV INFECTION, AIDS, AND CERVICAL CANCER.[11] T. VAGINALIS HAS ALSO BEEN REPORTED IN THE URINARY TRACT, FALLOPIAN TUBES, AND PELVIS AND CAN CAUSE PNEUMONIA, BRONCHITIS, AND ORAL LESIONS. CONDOMS ARE EFFECTIVE AT REDUCING, BUT NOT WHOLLY PREVENTING, TRANSMISSION.[12]
- TRICHOMONAS VAGINALIS INFECTION IN MALES HAS BEEN FOUND TO CAUSE ASYMPTOMATIC URETHRITIS AND PROSTATITIS.[13] IT HAS BEEN PROPOSED THAT IT MAY INCREASE THE RISK OF PROSTATE CANCER; HOWEVER, EVIDENCE IS INSUFFICIENT TO SUPPORT THIS ASSOCIATION AS OF 2014.

LIFE CYCLE

- TRICHOMONAS VAGINALIS RESIDES IN THE FEMALE LOWER GENITAL TRACT AND THE MALE URETHRA AND PROSTATE
- WHERE IT REPLICATES BY BINARY FISSION
- THE PARASITE DOES NOT APPEAR TO HAVE A CYST FORM, AND DOES NOT SURVIVE WELL IN THE EXTERNAL ENVIRONMENT.
- TRICHOMONAS VAGINALIS IS TRANSMITTED AMONG HUMANS, ITS ONLY KNOWN HOST, PRIMARILY BY SEXUAL INTERCOURSE


DPDx
Nonpathogenic Flagellates
CDC
2a
Cysts ingested
2b
Trophozoites ingested
Enteromonas hominis
Retortamonas intestinalis
Pentatrichomonas hominis
TROPHOZOITES
CYSTS
Binucleate
Quadrinucleate
Enteromonas hominis
Retortamonas intestinalis
Pentatrichomonas hominis
(No cyst stage)
Noninvasive colonization
1
Cysts and/or trophozoites passed in feces.
Infective stage
Diagnostic stage

GENETIC DIVERSITY

RECENT STUDIES INTO THE GENETIC DIVERSITY OF T.VAGINALIS HAS SHOWN THAT THERE ARE TWO DISTINCT LINEAGES OF THE PARASITE FOUND WORLDWIDE; BOTH LINEAGES ARE REPRESENTED EVENLY IN FIELD ISOLATES. THE TWO LINEAGES DIFFER IN WHETHER OR NOT T.VAGINALIS VIRUS (TVV) INFECTION IS PRESENT. TVV INFECTION IN T.VAGINALIS IS CLINICALLY RELEVANT IN THAT, WHEN PRESENT, TVV HAS AN EFFECT ON PARASITE RESISTANCE TO METRONIDAZOLE, A FIRST LINE DRUG TREATMENT FOR HUMAN TRICHOMONIASIS.

GENOME SEQUENCING AND STATISTICS

The *T. vaginalis* genome was found to be approximately 160 megabases in size[26] – ten times larger than predicted from earlier gel-based chromosome sizing.[27] (The human genome is ~3.5 gigabases by comparison.[28]) As much as two-thirds of the *T. vaginalis* sequence consists of repetitive and transposable elements, reflecting a massive, evolutionarily recent expansion of the genome. The total number of predicted protein-coding genes is ~98,000, which includes ~38,000 'repeat' genes (virus-like, transposon-like, retrotransposon-like, and unclassified repeats, all with high copy number and low polymorphism). Approximately 26,000 of the protein-coding genes have been classed as 'evidence-supported' (similar either to known proteins, or to ESTs), while the remainder have no known function. These extraordinary genome statistics are likely to change downward as the genome sequence, currently very fragmented due to the difficulty of ordering repetitive DNA, is assembled into chromosomes, and as more transcription data (ESTs, microarrays) accumulate. But it appears that the gene number of the single-celled parasite *T. vaginalis* is, at minimum, on par with that of its host *H. sapiens*.

In late 2007 TrichDB.org was launched as a free, public genomic data repository and retrieval service devoted to genome-scale trichomonad data. The site currently contains all of the *T. vaginalis* sequence project data, several EST libraries, and tools for data mining and display. TrichDB is part of the NIH/NIAID-funded EupathDB functional genomics database project

Virulence factors

- One of the hallmark features of Trichomonas vaginalis is the adherence factors that allow cervicovaginal epithelium colonization in women. The adherence that this organism illustrates is specific to vaginal epithelial cells (VECs) being pH, time and temperature dependent. A variety of virulence factors mediate this process some of which are the microtubules, microfilaments, bacterial adhesins (4), and cysteine proteinases. The adhesins are four trichomonad enzymes called AP65, AP51, AP33, and AP23 that mediate the interaction of the parasite to the receptor molecules on VECs.[24] Cysteine proteinases may be another virulence factor because not only do these 30 kDa proteins bind to host cell surfaces but also may degrade extracellular matrix proteins like hemoglobin, fibronectin or collagen IV.

Protein function

- Trichomonas vaginalis lacks mitochondria and therefore necessary enzymes and cytochromes to conduct oxidative phosphorylation. T. vaginalis obtains nutrients by transport through the cell membrane and by phagocytosis. The organism is able to maintain energy requirements by the use of a small amount of enzymes to provide energy via glycolysis of glucose to glycerol and succinate in the cytoplasm, followed by further conversion of pyruvate and malate to hydrogen and acetate in an organelle called the hydrogenosome.

Increased susceptibility to HIV

- The damage caused by Trichomonas vaginalis to the vaginal epithelium increases a woman's susceptibility to an HIV infection. In addition to inflammation, the parasite also causes lysis of epithelial cells and RBCs in the area leading to more inflammation and disruption of the protective barrier usually provided by the epithelium. Having Trichomonas vaginalis also may increase the chances of the infected woman transmitting HIV to her sexual partner(s).

DIAGNOSIS

CLASSICALLY, WITH A CERVICAL SMEAR, INFECTED WOMEN MAY HAVE A TRANSPARENT "HALO" AROUND THEIR SUPERFICIAL CELL NUCLEUS BUT MORE TYPICALLY THE ORGANISM ITSELF IS SEEN WITH A SLIGHT CYANOPHILIC TINGE, FAINT ECCENTRIC NUCLEI, AND FINE ACIDOPHILIC GRANULES.[14] IT IS UNRELIABLY DETECTED BY STUDYING A GENITAL DISCHARGE OR WITH A CERVICAL SMEAR BECAUSE OF THEIR LOW SENSITIVITY. T. VAGINALIS WAS TRADITIONALLY DIAGNOSED VIA A WET MOUNT, IN WHICH "CORKSCREW" MOTILITY WAS OBSERVED. CURRENTLY, THE MOST COMMON METHOD OF DIAGNOSIS IS VIA OVERNIGHT CULTURE,[15][16] WITH A SENSITIVITY RANGE OF 75–95%.[17] NEWER METHODS, SUCH AS RAPID ANTIGEN TESTING AND TRANSCRIPTION-MEDIATED AMPLIFICATION, HAVE EVEN GREATER SENSITIVITY, BUT ARE NOT IN WIDESPREAD USE.[17] THE PRESENCE OF T. VAGINALIS CAN ALSO BE DIAGNOSED BY PCR, USING PRIMERS SPECIFIC FOR GENBANK

Treatment

- Infection is treated and cured with metronidazole[19] or tinidazole. The CDC recommends a one time dose of 2 grams of either metronidazole or tinidazole as the first-line treatment; the alternative treatment recommended is 500 milligrams of metronidazole, twice daily, for seven days if there is failure of the single-dose regimen.[20] Medication should be prescribed to any sexual partner(s) as well because they may be asymptomatic carriers.

FOR BETTER UNDERSTANDING

- https://www.youtube.com/watch?v=SYd4lLed3CI
- https://www.youtube.com/watch?v=yk0P7IpSilg
- https://www.youtube.com/watch?v=TlNBQx9rH20&list=TLPQMTMwNjIwMjBRQCLpCuaiHQ&index=3

THANK YOU