

Rank: _____

Virginia Regional Science Olympiad Tournament

Microbe Mission Division C

Team #: ___KEY___

School: _____

Rules of Engagement:

- Do not open the test before the proctor has given you permission to do so.
- You will have a maximum 50 minutes to complete the event; you may leave before the event is complete if you have finished your event examination before the time has expired, but you will *not* be allowed to return for any reason until after the event has ended.
- You may separate the pages for this examination to divide the test-taking duties, but please return the pages back in the original order; also please write your team number at the top right hand corner of each page to allow the proctor(s) to associate the tests correctly to your team if the pages are inadvertently separated later.
- *Where applicable*, short, simple, bullet-point answers and appropriately-labeled diagrams are acceptable responses; long, expository essays are unnecessary, especially for short answer responses; if space is limited, you may use the back – *label/number your responses* so we can give due credit for answers.
- A word of encouragement – this event is designed to challenge you, so do not be worried if you feel you are doing poorly; chances are, your neighbors are feeling the same way as well.



“Be careful - they sense fear.”

Score: _____

Team: _____

Part A – *Fungi*

1. To the right is an image of *Saccharomyces cerevisiae*. The cell wall has been colored using false stains. In particular, the yellow sections (budding) indicate scarring of the cell wall. These scars are evidence of what cellular process? (1)

mitosis

2. What microscope was most likely used to generate this image? (1)

SEM

3. The image to the right highlights another aspect of the *S. cerevisiae* life cycle – the four reproductive cells enclosed in a cellular sac. What are the names of the sac and of the reproductive bodies inside it? (2 *T*)

ascus (ascospores)

4. Which process of cellular reproduction is used to generate this structure? (1)

meiosis

5. The diagram to the right shows the *S. cerevisiae* life cycle more simply. What is the name of the union at #2? (1)

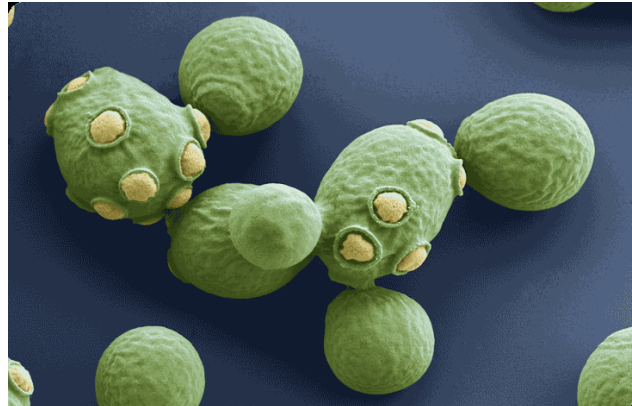
Plasmogamy (fusion of cell membrane)

6. Unlike many fungi, *S. cerevisiae* does not have an extensive dikaryotic life cycle stage. What is meant when a fungal organism is “dikaryotic” and what step of the fungal life cycle ends the dikaryotic stage? (2)

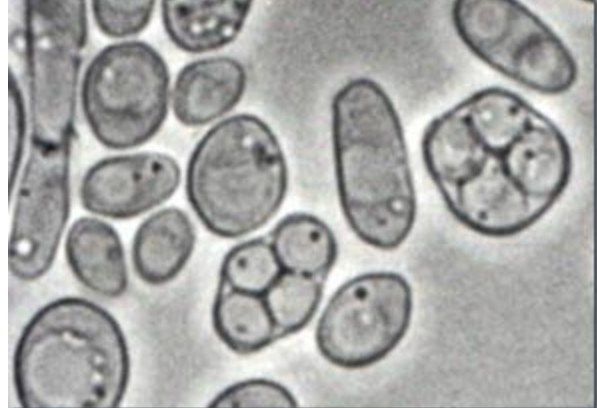
Plasmogamy (fusion of cell membranes) has occurred, but karyogamy (fusion of nuclei) has not occurred yet.

7. This question is asked every year, and no one ever answers this one correctly. To the right is a *Neospora crassa* hyphal tip, with a brightly stained organelle (arrow pointed at it!) without any membranes. What is the name of this fungal organelle? (1 *T*)

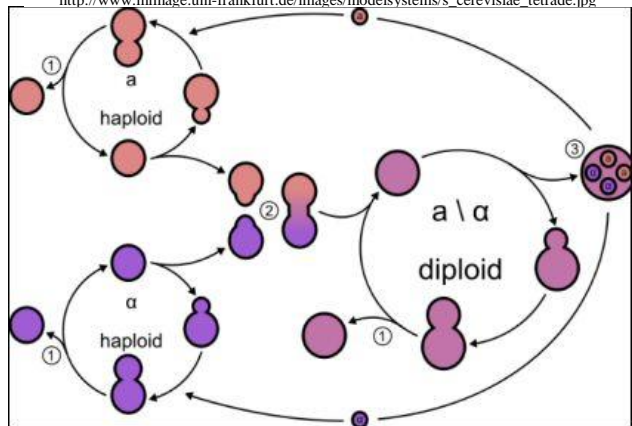
Spitzenkorper



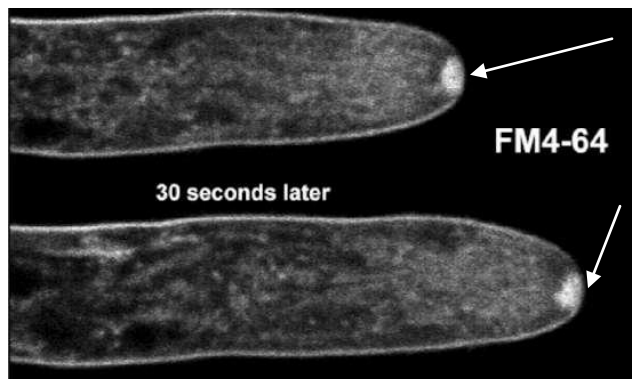
http://www.microbiologyonline.org.uk/themed/sgm/img/slideshows/3.1.4_fungi_2.png



http://www.mimage.uni-frankfurt.de/images/modelsystems/s_cerevisiae_tetrad.jpg



<http://www.wynboer.co.za/imagesart/biotech.jpg>



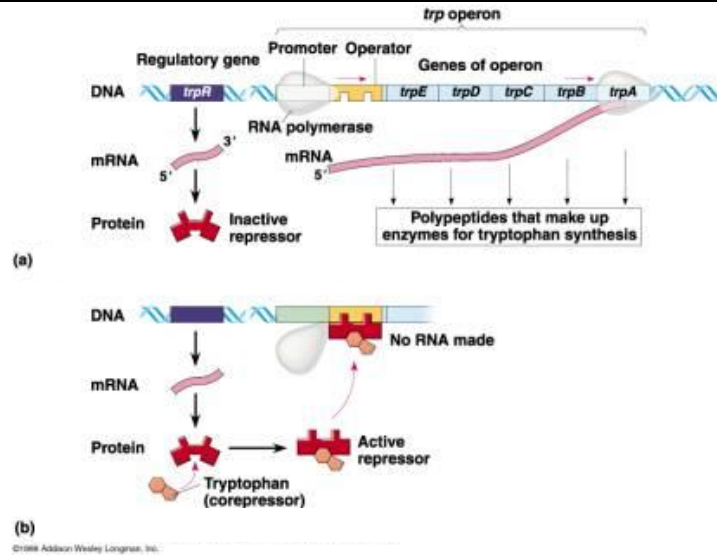
http://www.biology.ed.ac.uk/research/groups/jdeacon/FungalBiology/Fig3_15a.jpg

Team: _____

Part B – Bacteria

1. Lacking the complexity of gene splicing in eukaryotes, many bacterial proteins are synthesized in groups of closely related genes (especially those involved in metabolism) called *operons*. What is the primary function of an operon? (2)

Operons allow bacteria to regulate expression of multiple genes using relatively simple gene switches.



http://departments.oxy.edu/biology/bio130/lectures_2000/11-17-00_lecture_files/image022.jpg

2. The diagram above illustrates the activity of the *trp* operon, which governs tryptophan biosynthesis in *E. coli*. According to this diagram, if excess tryptophan is present, is gene expression activated or repressed?

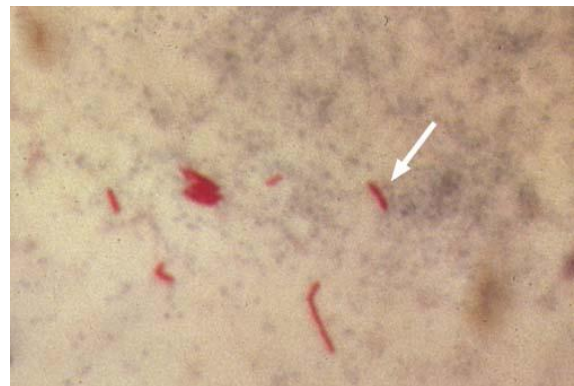
3. The above diagram shows an example of which of the following?

- a. feedback inhibition
- b. attenuation

- c. discontinuous protein synthesis
- d. competitive regulation

4. The diagram to the right shows a stained *Mycobacterium* infection. How can we visualize the *Mycobacterium* cells to the right? (2 *T*)

Mycobacterium is a gram-indeterminate bacteria, meaning its cell wall does not react well to gram staining. To visualize this bacteria, researchers must use an acid fast stain. Despite this, *Mycobacteria* is in fact *gram-positive*.



<http://textbookofbacteriology.net/acid-fastbacilli.jpeg>

5. *Mycobacterium* is commonly associated with a disease of the lungs, causing scarring of the tissues that can be visualized on an X-ray scan. What is the common name of this disease?

Pneumonia

6. How would you describe the shape of the bacteria?

Bacillus

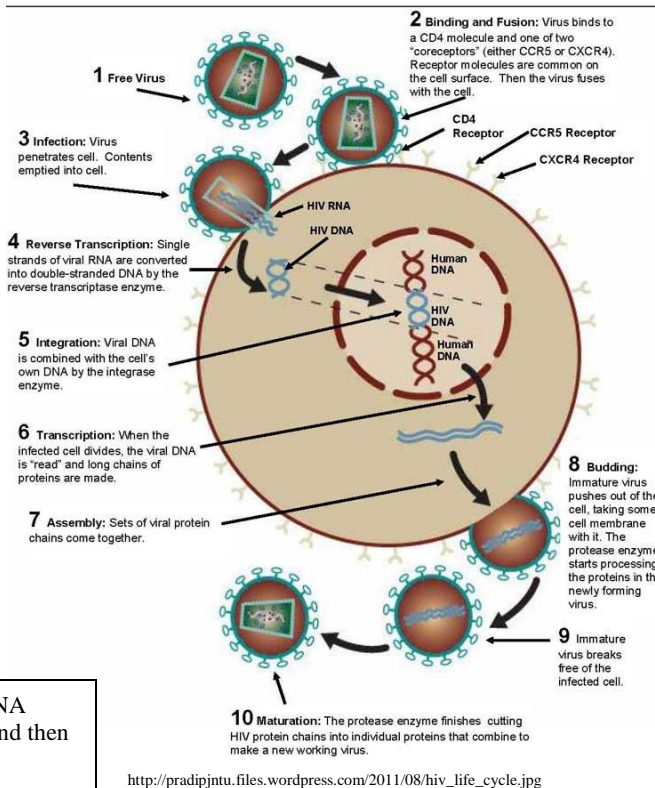
Team: _____

Part C – Viruses

HIV LIFE CYCLE



<http://trialx.com/curetalk/wp-content/blogs.dir/7/files/2011/05/diseases/Hiv-1-3.jpg>



1. What type of microscope most likely captured the image above?

TEM

2. HIV is classified as a *Group VI* virus. What does this mean? (2 *T*)

Group VI viruses are retroviruses; retroviruses have an RNA genome, but use RTase to transcribe genome into DNA, and then integrate into host genome.

3. A *very* recent paper in Nature (Feb 2012) described a new protein that shows anti-HIV action by depleting cells of the building blocks to produce DNA. Explain how this protein performs “anti-HIV” action, and why it would *not* be a viable treatment for HIV patients. (4)

Depleting building blocks of DNA would prevent the virus’s ability to perform RT and integrate into the host genome, since its RNA genome needs to be transcribed into DNA. This would prevent infection before the virus has been able to hijack the cellular machinery. However, since most human cells need to replicate DNA or perform DNA repair, using a modification of this protein would not be a good treatment.

4. Some current HIV treatments focus on the action of HIV-1 protease. What role does this protein fulfill in HIV replication? (2)

HIV-1 protease cleaves the polyprotein complex which is necessary for repackaging of viral proteins during replication and budding. Without proper cleaving of the polyprotein complex, the virus cannot erupt from the host cell, arresting replication.

5. HIV is a lytic cycle virus. What is the *other* lifestyle for viruses, and how does that lifestyle differ from that in lytic cycle viruses? (2)

Lysogenic viruses differ from lytic cycle viruses by the time latency between infection and lysis (or budding) of viral progeny. Whereas lytic viruses immediately begin producing new viral particles after infecting a host and integrating into the host genome, lysogenic viruses can lie in dormancy after infection and integration, even to the extent where the host cell has undergone several normal cell divisions. After an external stimulus triggers a return to the viral lifestyle, the lysogenic virus emerges from dormancy and resumes its work like a lytic virus.

