Birla Institute of Technology and Science, Pilani
Pharmaceutical Microbiology (PHA F217)
Second Semester 2020-21; Comprehensive Examination

Time: 90 Min | **40 Marks : Closed Book** | Date: 07/05/2022

Q1. Why people with HSV-1 are more prone to Alzheimer's disease? What happens during Latent Syphilis? **[3]**

Q2. Discuss the sequence of infection of the intestinal wall by Shigella. What is the effect of macrophages on Shigella infection? **[3]**

Q3. What is an obligate intracellular parasite? Discuss about their survival strategies. **[1+3]**

Q4. What is the viral envelope, and what does the presence (or absence) of it tell you about the type of virus? How does the viral envelope both aid in cell entry as well as hide it from being detected by the immune system? **[2+3]**

Q5. Explain our current understanding of molecular adaptations to the cytoplasmic membrane that are present in psychrophiles and describe their habitat. How about thermophiles? **[2+2]**

Q6. A patient has been diagnosed as having diarrhea. Is this sufficient information to begin treatment with antimicrobial agents? Briefly discuss why or why not. **[3]**

Q7. **a.** Pick an antimicrobial drug and describe its mechanism of action. **[3]**

b. Describe 2 separate mechanisms by which bacteria could develop resistance to this drug. **[4]**

c. Describe a strategy that could be used to augment and/or modify your original drug to make it effective against bacteria that have acquired resistance. **[3]**

Q8. You have a new position at the Center for Disease Control. You have been assigned to characterize a new epidemic that has spread rapidly among workers at a chicken farm in California. All affected have high fever and a severe cough. A co-worker has given you a purified vial of the infectious material.

a) You add some of the infectious material to an agar plate containing all necessary nutrients, but nothing grows. Why is this? **[2]**

b) You then add infectious material to a human cell line and see some cells begin to round up and die. What is the likely infectious material and why didn't it grow on the agar plate? If the cells were put into a tube with the media they grew in and spun down, where would you find the material that could infect new cells? In the supernatant or in the pelleted cells? Why? **[1+3]**

c) You isolate the genetic material of the pathogen and send a portion off for sequencing. Your sequence contains Uracil instead of Thymidine and it seems it is a single stranded genome. Can you identify the pathogen? **[2]**

★★★★★★★★★ Best Wishes ★★★★★★★★★

Birla Institute of Technology and Science, Pilani
Pharmaceutical Microbiology (PHA F217)
Second Semester 2020-21; Comprehensive Examination

Time: 90 Min | **40 Marks : Open Book** | Date: 07/05/2022

Q1. Describe how the cell wall component of any bacteria impact its pathogenicity and treatment. **[3]**

Q2. The following description is for MacConkey sorbital (SMAC) agar:
Mode of Action:
Bile salts mixture and crystal violet largely inhibit the growth of the Gram-positive microbial flora. Sorbitol, together with the pH indicator neutral red, is used to detect sorbitol-positive colonies and turning them red in color. Sorbitol-negative strains, on the other hand, form colorless colonies. Is this a selective or a differential medium? Most commensal E. coli strains ferment sorbitol, while pathogenic O157:H7 enterohemmorhagic E. coli (EHEC) do not. What color do you expect each of these colonies to be on SMAC plates? **[2+3]**

Q3. Exactly 100 bacteria with a generation time of 30 minutes are introduced into fresh sterile broth at 8:00 A.M. and maintained at an optimum incubation temperature throughout the day. How many bacteria are present at 3:00 P.M.? How many generations will take place by 5:00 P.M.? **[3]**

Q4. How do microbial communities function to affect the phenotype of the host? **[3]**

Q5. Shiga toxin (Stx) is a potent A-B toxin that inhibits protein synthesis and has a LD50 of approximately 20 ng/kg of body weight in rabbits. Nonetheless, cell lines derived from different mammalian tissues range from highly susceptible (cytotoxic dose 50 [CD50] of approximately 10 pg/ml) to completely resistant (CD50 > 1 μg/ml). What cellular or molecular differences do you think account for susceptibility and resistance of these cell lines? How could you test your hypothesis? **[3+5]**

Q6. What role does the common cold have in the rise of antibiotic-resistant strains of bacteria? Explain your reasoning. **[3]**

Q7. Suppose you are a medical technician working in a hospital laboratory and you collected a sputum sample from a critically ill patient suspected of having tuberculosis. You of course go back to the lab and attempt to culture and identify this dangerous, fastidious, and slow-growing microorganism, a process that can take 8 weeks. However, a quick verification of the tuberculosis diagnosis is needed. Can you think of a possible method or technology that can help speed the process? If so, what is it and how does it work? **[5]**

Q8. Imagine that you are a scientist working for the local health department. Your microbiology laboratory has recently received several cultures of different Gram-negative enteric bacteria, Salmonella typhimurium, Salmonella enteriditis, Escherichia coli, and Shigella species that are all resistant to the same four different antibiotics. What could explain how all these different bacteria acquired resistance to the same four antibiotics? What would you look for to confirm your hypothesis? **[2+3]**

Q9. Assume that you are a microbiologist who has been doing research on a penicillin-sensitive strain of Staphylococcus aureus for many months. One day you discover that the organism is now resistant to penicillin. You know that it has not come in contact with any other species of bacteria, nor has it come in contact with the DNA from any other species of bacteria. What are the two possible explanations for its sudden change from penicillin susceptibility to penicillin resistance? **[5]**

★★★★★★★★★ Best Wishes ★★★★★★★★★