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as they are not specific for C. pneumoniae but identify the chlamydiae only to the genus level. TREATMENT C. pneumoniae Infections Although few controlled trials of treatment have been reported, C. pneumoniae is inhibited in vitro by erythromycin, tetracy cline, azithromycin, clarithromycin, gatifloxacin, and gemifloxacin. Directed therapies include azithromycin 500 mg orally once fol lowed by 250 mg on days 2–5; doxycycline 100 mg orally twice daily; or clarithromycin 500 mg twice daily. The fluoroquinolones levofloxacin (750 mg orally once daily) and moxifloxacin (400 mg orally once daily) are alternatives, but their role is limited by increasing safety concerns due to serious side effects. For most patients, a 5-day course of therapy is sufficient. Beta-lactams and trimethoprim/sulfamethoxazole are not active. Acknowledgment The authors wish to thank Dr. Charlotte A. Gaydos for her contributions to this chapter in previous editions. ■ ■FURTHER READING Centers for Disease Control and Prevention: Sexually Trans mitted Infections Surveillance, 2022. Atlanta, GA: U.S. Department of Health and Human Services, 2024. https://www.cdc.gov/std/statistics/2022/ default.htm. Centers for Disease Control and Prevention: Sexually trans mitted infections treatment guidelines, 2021. MMWR Recomm Rep 70:1, 2021. Elwell C et al: Chlamydia cell biology and pathogenesis. Nat Rev Microbiol 14:385, 2016. Gaydos CA, Essiq A: Chlamydiaceae, in Manual of Clinical Microbiol ogy, 11th ed. JH Jorgensen et al (eds). Washington, DC, ASM Press, 2015, pp 1106–1121. Goller JL et al: Population attributable fraction of pelvic inflamma tory disease associated with chlamydia and gonorrhoea: A crosssectional analysis of Australian sexual health clinic data. Sex Transm Infect 92:525, 2016. Gregory ECW, Ely DM: Trends and characteristics of sexually transmitted infections during pregnancy: United States, 2016–2018. National Vital Statistics Reports 69:1, 2020. Hammerschlag MR et al: Chlamydia pneumoniae, in Mandell, Doug las, and Bennett’s Principles and Practice of Infectious Diseases, 9th ed. JE Bennett, R Dolin, MJ Blaser (eds). Philadelphia, Elsevier, 2020, Chapter 182. Hughes Y et al: Universal lymphogranuloma venereum (LGV) testing of rectal chlamydia in men who have sex with men and detection of asymptomatic LGV. Sex Transm Infect 98:582, 2022. Kuypers J et al:

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Section 11 Viral Diseases: General Considerations David M. Knipe, Max L. Nibert

Principles of Medical

Virology Viruses are obligate intracellular parasites that must enter cells to replicate and propagate themselves to spread to other cells. Infection often injures the host cell—hence the name “virus,” derived from the Latin word virus for poison or toxin. Viruses are one of the simplest life forms and, at the minimum, have a nucleic acid genome with a protein coat. They do not divide by division, as do cells; instead, viruses are programmed to disassemble inside cells, to use their nucleic acid genome to encode viral proteins that replicate their genomic nucleic acid, and then to assemble the progeny genomes into viral particles. The progeny viruses are secreted or released from the host cell as extra cellular virions that infect surrounding cells. Viruses depend on the host cell for many of the enzymes and organelles that synthesize carbo hydrates, lipids, nucleic precursors and nucleic acids, and high-energy molecules, including the host cell’s ribosomes, which are used to make viral proteins. In the process of taking over the host cell, viruses inhibit normal cell metabolic pathways and cause damage to the cell in a pro cess that results in the cytopathic effect (CPE). Injury to cells and cell death can cause tissue damage and contribute to virus-induced disease. CHAPTER 195 Viruses are distinct from other intracellular parasites such as viroids, virusoids, prions, and intracellular bacteria. Viroids are small, circular, single-stranded RNA infectious pathogens of plants that do not have a protein coat, while virusoids are small, circular-RNA, infectious patho gens that depend on viruses to provide the proteins for their replica tion and protein coat. Prions are misfolded proteins that spread from one cell to another, causing the same protein molecules to misfold in the new cell. The misfolded proteins in prions cause cellular damage (Chap. 449). Principles of Medical Virology VIRUS STRUCTURE There are many different virus structures, but nearly all are formed from a few fundamental structural elements. The minimal virion particle is composed of a complex of nucleic acids (the genome) and a protein shell (the capsid) (Fig. 195-1). The combination of the genome and the capsid is called the nucleocapsid. The genome is protected within the capsid. The external surface of virions can consist of either the protein capsid or a lipid envelope around the capsid (Fig. 195-1). Viral genomes can consist of single- or double-stranded RNA or DNA and can comprise one or more genome segments. Singlestranded (ss) genomes are designated as positive strand (+) if they con tain the sequences encoding the open reading frames for viral proteins, while they are designated as negative strand (–) if they contain only complementary sequences. Thus, a positive-strand RNA viral genome can be translated into a viral protein upon entry into the host cell, while a negative-strand genome must be copied into complementary RNA molecules for translation. This dilemma is solved in negative-strand viruses by the loading of transcriptases onto the viral genome prior to

encapsidation; these enzymes transcribe the genome into viral mRNA upon entry into and uncoating within the cell. Viral capsids are made of repeating protein subunits because their genomes have limited coding capacity. The capsids are constructed with a few structural units or capsomers packed into a symmetrical arrangement. Capsids are usually organized in one of two ways: (1) an icosahedral or spherical symmetry based on an icosahedron with two-, three-, and fivefold axes of symmetry formed from 20 triangular faces or (2) a helical symmetry. However, viruses occasionally have more complex structures (e.g., the poxviruses) (Fig. 195-2).

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