Biology of Chlamydia Trachomatis

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경기대 생물학

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Chlamydiae are extremely successful pathogens of humans and animals. As obligate intracellular parasites, chlamydiae have evolved unique adaptations to intracellular parasitism.

Terminology

The genus is Chlamydia, but the trivial names "chlamydiae" (plural) and "chlamydia" (singular) are often used in addition to the adjective "chlamydial".

The two major developmental forms, the elementary body and the reticulate body, are referred to as the EB and the RB. Chlamydiae have a "developmental cycle", not a "life cycle" whose end would result in death or terminal differentiation. The intracellular vacuole in which chlamydiae grow is called an "inclusion".

Nomenclature and Taxonomy

Chlamydiae were originally thought to be protozoa and later viruses, but it became clear that chlamydiae had all the requisite properties of bacteria. Chlamydiae, have been placed in their own order, Chlamydiales, with one family, Chlamydiaceae, and a single genus, Chlamydia. The genus Chlamydia consists of three major species, Chlamydia trachomatis, C. psittaci, and a new species, C. pneumoniae. There is increasing acceptance of a species designation for a subset of C. psittaci called C. pecorum. C. trachomatis has been divided into three

biovariants (biovars): trachoma, lympho- granuloma venereum(LGV), and murine(mouse pneumonitis (MoPn) agent). The trachoma and LGV biovars appear to be essentially identical and the murine biovar is

more distantly related. The trachoma and LGV biovars are distinguished by significantly different clinical features. LGV biovar strains readily cause systemic infections and proliferate in lymph nodes, whereas growth of the trachoma biovar is believed to be limited to columnar epithelial cells at mucosal surfaces. The trachoma biovar consists of prototypical serovariants (serovars) designated by the letters A through K, including serovars Ba, Da, and Ia, The LGV biovar consists of four serovars, L1, L2, L2a, and L3.

Developmental Biology

The chlamydial developmental cycle is the conversion of EBs to RBs, the logarithmic division of RBs, and the reorganization of RBs back to EBs. The middle of the cycle presumably is much like that of most other organotrophic bacteria, except that growth and cell division are restricted to an intracellular environment., It is the beginning and the end of the cycle that are most interesting and make Chlamydia unique. The adaptation appears to be two-fold: resistance to physical and chemical pressures present in the extracellular world and a shutdown of metabolism.

Cell Biology

All intracellular parasites must accomplish several essential functions. They must have the ability to enter host cells, avoid cellular defense mechanisms, create an intracellular niche supportive of multiplication, exit the host cell, and survive extracellularly to invade subsequent host cells.

Attachment

The process of EB internalization is so efficient that it has been termed "parasite- specified endocytosis" to emphasize the belief that EBs trigger their own

internalization by cells. Although chlamydial infections are, for the most part, limited to mucosal surfaces and are usually quite specific in their host range, in culture they successfully enter and multiply within a wide variety of cell types from many different vertebrate species.

Entry

Mechanisms proposed to mediate chlamydial entry are as varied as the putative ligands mediating attachment. Because chlamydiae efficiently enter nonprofessional phagocytes, it can be expected that the initial interaction with an as yet unidentified host plasma membrane receptor activates signal transduction processes that stimulate a local rearrangement of the cytoskeleton to induce internalization of the EB. A zipper-type mechanism of chlamydial entry has been proposed, as there is no morpho- logical evidence of activation of macropinocytosis during chlamydial entry.

Intracellular Development

Intracellular parasites have evolved diverse strategies for evasion of host cellular defense mechanisms associated with adaptations for survival in distinct intracellular compartments. Intracellular parasites that replicate within vacuoles are believed to arrest maturation of those vesicles at a stage appropriate to the physiology of the parasite.

Effects on the host cell

Chlamydial infection causes surprisingly little disruption of normal host cell functions. Cells infected with a low multiplicity of infection synthesize DNA and progress through the cell cycle with minimal disruption until late in the developmental cycle. Host protein synthesis is also only minimally affected by low multiplicities of chlamydial infection. Host glycolysis and respiration are stimulated in response to chlamydial infection.

Release

Chlamydia completes its intracellular development by 36h or longer. The more rapidly growing strains, primarily C. psittaci and the LGV strains of C. trachomatis, tend to be released by lysis of the host cell. However, certain

serovars of C. trachomatis do not lyse the host cell at the completion of replication but are released by a process in which the inclusion membrane fuses with the plasma membrane to release the contents of the inclusion to the environment. Functionally and topologically, this process is consistent with the view that the chlamydial inclusion represents an exocytic vesicle in which transport to and fusion with the plasma membrane are inhibited or delayed.