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Mycoplasma pneumoniae — its nature and factors involved
in colonisation and infection

Mycoplasma pneumoniae — charakter drobnoustroju i cechy związane z kolonizacją
i zakażeniem

Mycoplasmas are included within the class *Mollicutes* (“soft skin”), which comprises four orders, five families, eight genera and more than 150 species. Mollicutes are eubacteria which have evolved from clostridia-like gram-positive cells by gene deletion. The permanent lack of a cell wall barrier makes the mycoplasmas unique among prokaryotes and differentiates them from bacterial L forms, for which the lack of the cell wall is a temporary reflection of environmental conditions. The lack of the cell wall also renders those organisms insensitive to the activities of β -lactam antibiotics, prevents them from staining by the Gram technique and is responsible for their pleomorphical form. The extremely small genome, only around 1 Mb, and limited biosynthetic capabilities explain the parasitic or saprophytic existence, their sensitivity to environmental conditions and their fastidious growth requirements [8]. Although mycoplasmas lack many metabolic pathways and peptidoglycans layers, they have advanced system required for parasitic life, i.e., internalisation in host cells, membrane transport, antigenic variations, host cell adhesion, gliding motility [9]. Cell reproduction cycle of mycoplasma has been recently partially elucidated with reference to DNA replication, cell division and formation of attachment organelle. Study of attachment organelle can yield clues to the cell reproduction cycle of mycoplasmas because its formation and migration appear to couple with cell cycle. *Mycoplasma pneumoniae* attaches to ciliated epithelial cells by specialised terminal organelle. Several proteins have been identified to be related to the attachment organelle to *Mycoplasma pneumoniae*. P1 protein is located on the membrane at the attachment organelle and has direct roles in adhesion. A few others proteins are located at the attachment organelle and work as accessory proteins essential for cell adhesion [7].

In humans, mycoplasmas and ureaplasmas are mucosally associated, residing predominantly in the respiratory and urogenital tracts and rarely penetrating the submu-

cosa, except the cases of immunocompromised patients. Instrumentation during hospital stay lets them invade the bloodstream and dissemination to many different organs and tissues. Many mycoplasma species exist as commensal organisms in the oropharynx, urogenital tract, the throat and other body sites including joints. *Mycoplasma pneumoniae* is a common cause of upper and lower respiratory tract infections of varied severity, causes approximately 20% of all community-acquired pneumonias which has been shown to occur endemically or epidemically in repeating cycle four to five years. Asymptomatic carriage of *Mycoplasma pneumoniae* as well as nosocomial pneumonia following postoperative assisted ventilation have been reported [2]. After inhalation of infected material, *Mycoplasma pneumoniae* binds to respiratory epithelial cells and induces inflammation. Locally produced secretory IgA may inhibit the binding to respiratory epithelium and those antibodies seem to play a greater role than serum antibodies in the protection against repeated mycoplasmal infections. In fatal cases of mycoplasmal pneumonia microorganisms are rarely demonstrated in lung tissue. In immunocompromised patients with severe mycoplasmal infection the chest radiographic changes were minimal or absent. That could be explained by decreased immunological reactivity in those patients, and therefore it would support the assumption that immunological mechanisms play a pathogenic role [1]. *Mycoplasma pneumoniae* is also responsible for producing a wide spectrum of nonpulmonary manifestations including neurological, hepatic, cardiac, and haematological diseases. The neurological manifestations are reported to be the most common nonpulmonary manifestation [13]. Association of Mycoplasma infection with asthma has been studied in many ways. Retrospective studies of serological findings revealed that recent mycoplasma infection may precede asthma onset, exacerbation of asthma and occurrence of status asthmaticus [5]. An acute exacerbation of wheezing in asthmatic participants in association with Mycoplasma infection has been also documented [4]. Several studies have shown that production of IgE specific to Mycoplasma is the results of changed balance of T helper type 1 (TH1)/T helper type 2 (TH2) regulating immune response and IgE synthesis [6].

SEROLOGICAL METHODS FOR DIAGNOSIS OF *MYCOPLASMA PNEUMONIAE* INFECTIONS

Mycoplasma pneumoniae is a fastidious organism, and culture is available only in a few laboratories. Although isolation is difficult as well as slow to be clinically relevant, it is regarded as a diagnostic gold standard especially during the evaluation of serological methods [12]. Since that organism is not sensitive to β -lactam antibiotics which are often used for the empirical treatment of lower respiratory tract infections, a rapid diagnostic method is required to avoid the use ineffective antibiotics. As a routine in microbiological laboratories serology remains a common method for diagnosis of acute and chronic *Mycoplasma pneumoniae* infections. The primary methods in serology are: as the first developed but non-specific cold hemagglutinin test (CHT) that is not in use now at all, complement fixation test (CFT) commonly employed test, parti-

cle agglutination test (PAT), indirect hemagglutination test (IHT) and enzyme immunoassays test (EIAs and ELISA). The CHT was found to be positive in only 30–50% of *Mycoplasma* infections and on the other hand positive also in other respiratory infections, collagen-vascular diseases and myelomas. The sensitivity of CFT test varies from 64% to 90%, and measures predominantly “early IgM antibodies” thus being less suitable to chronic repeated infection. However, rise in antibody titre of more than four fold in paired serum samples is usually thought to be a good evidence of recent infection. IgM is a reliable indicator of recent infection, but since this antibody is produced less frequently during reinfection, a negative results does not exclude recent infection, especially in patients over the age of 45. Higher *Mycoplasma pneumoniae* IgA antibody titer seems to be reliable indicator of recent infection [11]. In that test chlorophorm-methanol glycolipid extracts is employed as antigen whereas P1-enriched antigen is used in EIA tests, that is that guarantee of sensitivity and specificity of EIA tests [14]. Recently, polymerase chain reaction (PCR) tests for the detection of *Mycoplasma pneumoniae* have been developed [3]. These techniques seem to be useful for rapid diagnosis and they have high sensitivity and specificity, but up till now, not many routine laboratories have adequate equipment for that method.

High frequency of *Mycoplasma pneumoniae* infections and mentioned above diagnostic difficulties induce to recommendation for empiric antibiotic treatment. Tetracycline and macrolides such as erythromycin and azithromycin are the antibiotics of choice for treating *Mycoplasma pneumoniae* infections, which are resistant to all penicilins, cephalosporins, and trimethoprim [10].

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STRESZCZENIE

Mycoplasma pneumoniae są unikalnymi drobnoustrojami z powodu stałej utraty ściany komórkowej, wyjątkowo małego genomu, zmniejszonych możliwości metabolicznych, szczególnych wymagań wzrostowych i wyspecjalizowanego systemu dla pasożytniczego trybu życia: to jest pochłanianie przez komórki gospodarza, transport błonowy, zmienność antygenowa, adhezja do komórek gospodarza. Wysoce wyspecjalizowane organelle służące do adhezji zbudowane są z systemu odpowiednich białek. *Mycoplasma pneumoniae* jest częstą przyczyną infekcji górnych i dolnych dróg oddechowych o różnym stopniu ciężkości i szerokim spektrum pozapłucnych objawów. Związek *Mycoplasma pneumoniae* z astmą jest rozpatrywany w aspekcie wywołania astmy, zaostrzeń i komplikacji przebiegu. Serologiczne badania diagnostyczne obejmują testy EIA i ELISA i badania PCR, jeżeli są one możliwe do wykonania.