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PROBLEMS AND STRATEGIES IN THE DETECTION OF *Mycoplasma hominis*

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Among the diseases that are transmitted mainly sexually, urogenital mycoplasmosis occupies one of the first places. Clinical manifestations of the disease caused by *M. hominis* are often similar to the symptoms of other diseases of the urogenital tract of bacterial, viral and other etiologies. Therefore, the diagnosis of urogenital mycoplasmosis caused by *M. hominis* faces a number of problems, despite the possibility of using genetic methods for direct determination of the DNA of this microorganism.

Aim. Comprehensive analysis of the features of *M. hominis* and modern methods to determine promising approaches in the diagnosis of urogenital mycoplasmosis caused by this microorganism.

Methods. Search and systematization of modern scientific data and results on the features of the detection of mycoplasmas in the literature, covering numerous databases, including PubMed, Web of Science, Scopus, Google Scholar and other relevant sources.

Results. A comprehensive analysis of the features of the morphology, genetics and mechanisms of interaction with the human body of *M. hominis* was carried out. The possibilities of modern methods for detecting this pathogen of urogenital mycoplasmosis are considered, including genetic, immunological, immunocytochemical, immunohistochemical methods, as well as immunochromatographic tests, lateral flow assays or rapid tests.

Conclusions. The complexity of detecting *M. hominis* in the process of diagnosing urogenital mycoplasmosis is due to the morphological and genetic features of the pathogen, various mechanisms of masking from the immune system, its presence in the human body normally and in combination with other pathogens. The most effective strategy for detecting mycoplasmas in the diagnosis of diseases is the simultaneous determination of their antigens in urogenital samples and antibodies in the blood, which can also be a guideline for improving or developing new complex test systems.

Key words: *M. hominis*, urogenital mycoplasmosis, polymerase chain reaction, enzyme-linked immunosorbent assay, antigens, antibodies, diagnostics.

In recent years, urogenital mycoplasmosis has emerged as one of the leading diseases primarily transmitted through sexual contact. The causative agents of the disease are the smallest prokaryotes — the Gram-negative bacteria *Mycoplasma genitalium*, *Mycoplasma hominis*, and *Ureaplasma urealyticum*. Among these, the first is considered a pathogen, while the others are opportunistic conditionally pathogens and may be present in the body as part of the normal flora. While the presence of *M. genitalium* is a clear indicator of disease, the involvement of the other two aforementioned mycoplasmas in the patient's condition remains a question [1, 2].

Among the conditions that lead to the activation of *M. hominis* and *U. urealyticum*, causing inflammation in the urogenital tract,

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are immunodeficiency, stress, hypothermia, pregnancy, intoxication, avitaminosis, and co-existing chronic diseases. The prevalence of the disease across the general population is reported to be 1.3% in developed countries and 3.9% in developing countries [3, 4]. However, the WHO estimates that the carriage of these pathogens exceeds 20% of the population, and the diagnosis of urogenital mycoplasmosis must include the identification of each of them. Thus, according to various estimates, 21–54% of women, predominantly in younger age groups, and 4–13% of men are carriers of *M. hominis* [5, 6]. Asymptomatic carriage can progress to a clinically significant condition, a transition which, in Ukraine during the war, is facilitated by chronic stress, insufficient access to medical services, a general increase in the risk of other diseases, and the difficulty in adhering to hygiene rules.*M. hominis* primarily infects the organs of the genitourinary system and causes various destructive-inflammatory processes. In men, *M. hominis* typically causes urethritis and prostatitis, while in women, it leads to urethritis, cervicitis, and pelvic inflammatory disease (PID). This is particularly significant for women, as the consequences of *M. hominis* infection can include infertility and pregnancy complications. Urogenital mycoplasmosis in pregnant women can cause miscarriage, preterm birth, fetal infection, and the development of postpartum sepsis.

The clinical manifestations caused by the presence of *M. hominis* often resemble the symptoms of other urogenital tract diseases of bacterial, viral, or other etiologies. Therefore, the diagnosis of urogenital mycoplasmosis caused by *M. hominis* presents several challenges, despite the potential use of polymerase chain reaction (PCR) for the direct detection of the microorganism's DNA. Among these challenges is, first and foremost, the question of confirming the causative role of the detected (e.g., via PCR test) *M. hominis* in the disease, given that the microorganism is also present in the human body under normal conditions.

Consequently, the enzyme-linked immunosorbent assay (ELISA) method remains relevant in laboratory diagnostics. ELISA enables the determination of the body's reaction to the activity of *M. hominis* and is often faster, more convenient, and more economical to use. These method characteristics are significant, for example, in large-scale screening studies, when rapid analysis is needed, or when there is limited access to a laboratory for complex analyses (such as PCR).

In addition to the aforementioned primary methods for detecting infectious agents, other techniques are being developed, each with its own specific features and limitations in application. When factoring in the particular characteristics of the pathogen itself — where its presence is not always the cause of the disease — the issue of its detection and the selection of subsequent treatment strategies appears to be a task requiring comprehensive solutions.

Therefore, the aim of this work is to conduct a comprehensive analysis of the features of *M. hominis* and modern detection methods to determine prospective approaches in the diagnosis of urogenital mycoplasmosis caused by this microorganism.

Features of the Morphology, Genetics, and Host Interactions of *M. hominis*

A distinguishing feature of *M. hominis* bacteria is their small size (0.3–0.4 μm), the virtual absence of a cell wall, and a compact genome (700 kbp) [6, 7]. This makes it impossible to visualize bacteria under a light microscope and requires special conditions for in vitro cultivation in laboratory practice [8]. The bacterial membrane consists of a phospholipid bilayer, possessing a large number of integral and surface proteins, as well as lipoproteins. The structural nature of the protein component in these lipoproteins likely determines the differences in the mechanisms of mycoplasma immunomodulation compared to those of other bacteria. Surface proteins are the most crucial in the context of receptor recognition and host cell interaction mechanisms. Among all the lipids comprising the membrane, 35–50% are neutral (predominantly cholesterol), and 50–65% are polar [9].

The stability of the mycoplasma genome and protection against foreign DNA are maintained, in part, by the presence of restriction-modification systems [10]. However, the horizontal gene transfer between related genera is a common phenomenon due to their coexistence as parasites. Consequently, members of the Mollicutes class, such as the Mycoplasma and Ureaplasma species, share a standard set of genes. These genes are responsible for the metabolism of nucleotides, proteins, fatty acids, and phospholipids, as well as substrate transport. In contrast, species-specific traits are determined by

genes associated with energy metabolism, cytoadhesion mechanisms, and virulence. Even between pathovariants of the same species, differences in these characteristics are observed due to adverse selection [11, 1 2].

Species-specific genes and their expression products can be utilized for the identification of *M. hominis*. Species-specific genes and their expression products can be used for the identification of *M. hominis*. Mycoplasmas can exist both on the surface of the host cell membrane and intracellularly as «silent» parasites. Intracellular localization is sometimes observed in HIV-positive patients. Mycoplasmas infect various cell types — epithelial cells, endothelial cells, fibroblasts, and even immune cells. Among other members of the genus, *M. hominis* possesses the unique ability to penetrate the cytosol of spermatozoa and cause thinning of the midpiece; however, this mechanism does not apply to all cell types [13].

In the initial stage of mycoplasma interaction with host cells, lipoproteins and lipopeptides of the bacteria, as well as cellular receptors, play a vital role [14]. This recognition stage, in turn, triggers a cascade of events: rearrangements in cellular ultrastructure, reorganization of actin in the cytoskeleton, and activation of kinases. Consequently, mycoplasmas enter the cell via endocytosis [13]. Unlike other mycoplasmas, the surface proteins (adhesins) involved in these processes are not concentrated in a specialized cellular structure [15, 16]. The role of these proteins in cytoadhesion was investigated using the method of inhibition by monoclonal antibodies on HeLa cells [17, 18]. The lipids and lipoproteins of the mycoplasma cell membrane can trigger an inflammatory reaction and cellular damage through various mechanisms [19].

The adhesin P50 (or Vaa) — a protein ranging in size from 28 to 72 kDa [17] with an elongated C-terminal region and a globular N-terminal region — can exist in a truncated form (P50t), which significantly reduces the mycoplasma's ability to adhere. P50t is capable of macrophage adhesion and triggers an immune response via the TLR2 receptor [9]. Another essential adhesin, P80, contains a cytoplasmic signal peptide. Following enzymatic cleavage by signal peptidase-1, P80 is released from the membrane and can be recognized by the immune system [18].

The adhesin P120 (120 kDa) has a hypervariable region and undergoes lipid modification immediately before its incorporation into the membrane. Due to the presence of a hydrophobic region, it is soluble in both the lipid and aqueous phases [20]. P120 also has a homolog, P120', which does not induce an immune response. *M. hominis* also possesses a gene cluster, lmp1–lmp4, the products of which include two identified membrane proteins, LMP1 and LMP2, which vary in size and represent chimeric proteins [21].

Membrane-associated phospholipases and aminopeptidases may cause cellular damage during the adhesion process [22]. One such enzyme (OppA) is the substrate-binding domain of the oligonucleotide permease system. The adhesion process, mediated by OppA, also depends on the conformation of adjacent domains — CS1 and ATPase. Likely, one of the mechanisms by which OppA, as well as P50, interacts with cells is by binding to sulfoglycolipids on the cell surface [23,24].

The formation of exotoxins is generally atypical for mycoplasmas; however, their cytotoxicity is attributed to the secretion of H_2O_2, while the release of ammonia causes hyperammonemia syndrome and urolithiasis (kidney stones). *M. hominis* can also enhance the activity of *S. aureus* β-hemolysin via another virulence factor — CAMP [9]. *M. hominis* has also been shown to play a role in the deregulation of proteomic homeostasis by affecting the endoplasmic reticulum, deregulating the cell cycle, and interfering with apoptosis scenarios, proliferation processes, signaling pathways, and the pro-inflammatory response. Attempts have been made to identify proteins responsible for mitochondrial dysfunction. However, most of these studies were conducted on immortalized cell lines or through in silico design; therefore, convincing evidence that mycoplasmas are responsible for the development of any form of cancer has not been found [22, 25, 26].

From the perspective of host cells, the TLR2/6, TLR1/10, and TLR6 receptors play a key role in recognizing mycoplasma adhesins and developing the local immune response [15]. The heterodimers of these receptors recognize lipoproteins, causing the release of cytokines, as well as the activation of neutrophils and the release of secretory immunoglobulin A (sIgA). The humoral immune response to mycoplasmas is described by typical kinetics: specific class M antibodies are formed within one week of the onset of infection, followed by the appearance of specific IgG and IgA [27].

As with any pathogen, all components of the immune system are involved in

counteracting the infection during *M. hominis* pathogenesis, and the pathogen attempts to evade them. Among the mechanisms utilized by mycoplasmas to counteract the immune response are molecular mimicry, antigenic variation, resistance to oxidative stress, biofilm formation, degradation or evasion of neutrophil extracellular traps (NETs), and intracellular survival [13].

Antigenic variation is characteristic of surface proteins, particularly P120, LMP, and Vaa, which enable the pathogen to evade immune response mechanisms. This variation is also one of the challenges in developing a vaccine against *M. hominis*, although such attempts are ongoing [21, 28]. *M. hominis* also possesses a nuclease complex involved in the mechanism of evading NETs. One such enzyme, a Ca^{2+}-dependent nuclease (a surface lipoprotein), is capable of degrading both DNA and RNA. The degraded nucleic acid precursors can then be incorporated into the mycoplasma's metabolic processes [19].

Biofilm formation is not characteristic of all mycoplasmas, which is linked to their metabolism. The presence of arginine and thymidine in the environment can stimulate this process [19]. Biofilm formation makes the bacteria invisible to immune system cells and complicates subsequent treatment. Furthermore, *M. hominis* defends itself through intracellular parasitism, though this is sometimes associated not with host cells but with the cells of other pathogens. For example, a symbiotic relationship is known to occur between *M. hominis* and *T. vaginalis*, in which *T. vaginalis* functions as a protected niche for the mycoplasma, and treatment for trichomoniasis may trigger the release of *M. hominis* [29].

In addition to the "evasion" strategy, an interesting mechanism of mycoplasma defense is "confrontation" with immune system components by binding immunoglobulins or their complexes with specialized proteins: the immunoglobulin-binding protein (MIB) attaches to the light chain of the antibodies, and the immunoglobulin protease (MIP) connects to the newly formed MIB-IgG complex. In this state, it participates in the proteolytic cleavage of the heavy chain along with a serine protease, leading to the complete degradation of antibodies. Currently, there is no evidence of the functionality of this mechanism based on *in vitro* and in vivo studies for *M. hominis*; however, results from homology searches indicate the presence of this system in the bacterium [27].

Modern methods and strategies for detecting *M. hominis* in the diagnosis of urogenital mycoplasmosis

Modern methods for diagnosing urogenital mycoplasmosis include cultural, genetic, and immunological techniques for pathogen identification. These three testing modalities are widely used in laboratory practice, and diagnostic systems based on them have been broadly implemented in production. The feasibility and practical application of other methods, such as luminescent analysis or spectroscopy, are currently only being investigated.

The cultural method has historically been the first technique used for Mycoplasma diagnosis. A positive result from this test is irrefutable, and its significance in complex diagnostics has been proven by comparative studies [30]. The method is based on the cultivation of clinical urogenital samples (and less frequently, extragenital samples) in vitro using specialized media, followed by the identification of colonies. Mycoplasmas can be successfully detected at the stage of inflammation or colonization of the human urogenital tract. Two approaches are used in this diagnostic method: broth culture or growth on agar-based nutrient media.

The limitations of the cultural method for mycoplasmosis diagnosis include the duration of the procedure (from 24 hours), its low sensitivity compared to other methods (around 10^4 CFU/ml), and the risk of contamination despite the addition of selective agents. The method's advantages include its relative simplicity, thanks to the development of convenient commercial kits. Furthermore, it is currently the only approach that allows for the isolation of a pure culture and the determination of its antibiotic resistance [31].

Two traditional approaches are used for testing resistance to various antibiotics: broth and agar dilution. Modern variants of this testing can be implemented, for example, in the format of agar gradient diffusion (Etest, bioMérieux, France). In this test, strips containing gradient concentrations of antibiotics are inoculated onto agar plates, where antibiotic diffusion visually defines the zone where pathogen growth intersects the strip, which indicates the Minimum Inhibitory Concentration (MIC). [32]. Various commercial analogs of these tests are also available, which allow for the simultaneous identification of the pathogen and determination of resistance to a spectrum of antibiotics. These include:

Mycoplasma IST2, Mycoplasma IST3 (bioMérieux, France), Mycoplasma IES (Atlas Medical, UAE), the Mycofast revolution test series (ELITechGroup Microbiology Bruker Company, France), and Myco-Well-D-One (CPM SAS, Italy), which is designed for the simultaneous detection of *M. hominis* and *Ureaplasma spp.*. Despite utilizing traditional, long-established methods, these tests generally demonstrate acceptable diagnostic characteristics [33, 34].

Genetic methods are among the most widely used techniques today for diagnosing infectious diseases, including urogenital mycoplasmosis. The detection of mycoplasma nucleic acids using the polymerase chain reaction (PCR) method is characterized by high sensitivity and specificity, including the ability to differentiate between strains and pathovariants. This method is based on the enzymatic technique of amplifying species-specific nucleic acid fragments using specialized primers, which serve as sites for the elongation of the target DNA sequence. The procedure involves denaturation-replication cycles, which are controlled by temperature changes, allowing for the theoretical doubling of DNA quantity with each cycle (Fig. 1).

In the classical variant, the detection of the target gene is performed by agarose gel electrophoresis. However, the spread of the real-time PCR (qPCR) method allows for an immediate positive result due to the use of fluorogenic primer probes. In another qPCR variant, the fluorescent signal is directly proportional to DNA amplification, allowing for the quantitative assessment of DNA content [36]. The PCR method can also detect mycoplasmas in urogenital and extragenital samples during stages of acute inflammation or colonization of the urogenital tract. The quantitative determination of mycoplasmas can be helpful in evaluating the clinical picture, and real-time PCR (qPCR) enables the quantitative assessment of the sample using reaction kinetics data.

The most common genetic markers for bacterial identification are the 16S rRNA and 23S rRNA genes. This gene contains conserved regions suitable for PCR primer design, thus ensuring reliable and efficient amplification. Conversely, the presence of variable regions ensures effective differentiation between various species. Furthermore, it is not necessary to obtain a bacterial isolate to analyze this gene [37]. In cases where diagnosis is predicated on *M. hominis* as the causative agent, target genes may include known species-specific mycoplasma genes, such as gap, YidC, the unique amniotic fluid isolate genes goiB/goiC and alr, as well as genes encoding immunogenic structures like P120 or vaa [12, 38, 39].

Given the widespread nature of the method, a large number of PCR assays are available for detecting mycoplasmas or sexually transmitted infections (STIs). However, the majority of these are designed for research purposes or are intended to detect mycoplasmas in cell cultures, and thus are often not suitable for clinical diagnosis [38–41].

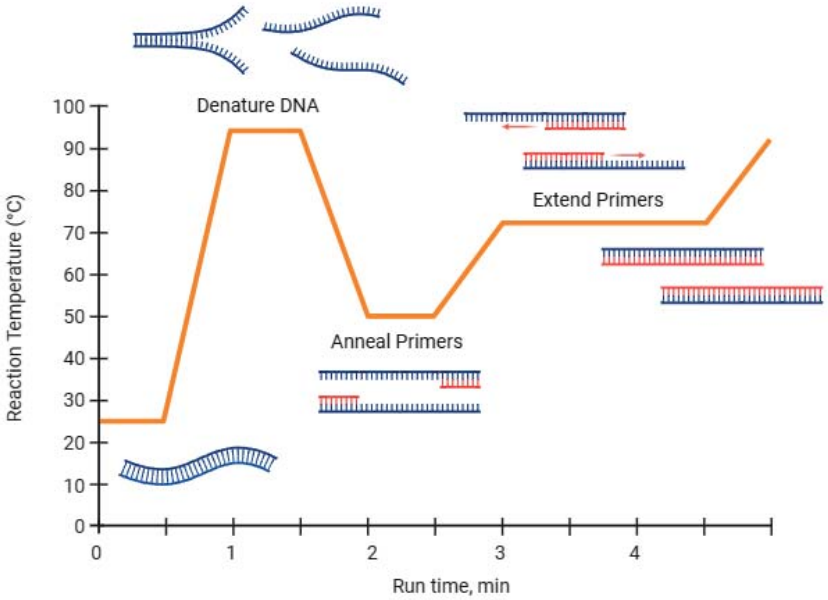

Fig. 1. **Illustration of the polymerase chain reaction [35]**

Examples of kits designed for use in diagnostics include the *Mycoplasma hominis* Real-Time PCR (BioPerfectus, China) and the *Mycoplasma hominis* PCR (Xema, Ukraine). The *Mycoplasma hominis* qPCR (NZYtech, Portugal) assay can detect at least 10 DNA copies per sample. A distinctive feature of this test is the DNA extraction control — an exogenous nucleic acid added to the lysis buffer to monitor for the presence of PCR inhibitors and the efficiency of DNA purification [42].

These tests are suitable for disease diagnosis that relies on the assumption of a specific pathogen. Conversely, to assess complex associations of microorganisms that collectively cause disease, multiplex PCR is utilized, which also allows for quantitative results based on reaction kinetics. Potential targets for the detection of *Mycoplasma* spp. Include the following genes: adh, mgpA, tetM, and tuf [43]. Examples of such medical devices include the *Mycoplasma genitalium/ hominis* test system (GeneProof, USA), which can detect both pathogens simultaneously by targeting the *M. hominis* gap gene and the *M. genitalium* 16S rRNA gene [44]. Another example is the sensitive and convenient EliGene® Mycoplasma hom/gen UNI test system (Elisabeth Pharmacon Ltd., Czech Republic), which detects 10 DNA copies with an analytical specificity of 100%. Its convenience stems from its compatibility with various platforms, including Roche, Applied Biosystems, Qiagen, ThermoFisher Scientific, Bio-Rad, and BioMolecular Systems [45].

Compared to the classical PCR procedure, the *isothermal amplification method* offers certain advantages. For instance, using only 4–6 primers targeting specific DNA regions, DNA can be rapidly amplified at lower temperatures (60–65 °C) and with less sophisticated equipment. Currently, this method has not yet found widespread use in diagnostics, and its primary application remains the screening of cell cultures [46]. For example, MycoStrip® (InvivoGen, USA) and Myco-Visible Mycoplasma Rapid Test Kit (MP Biomedicals, USA) consist of a set of reagents and a detection cassette. The prepared sample is incubated with reagents for the amplification of 16S rRNA and is then developed using an immunochromatographic strip [47, 48].

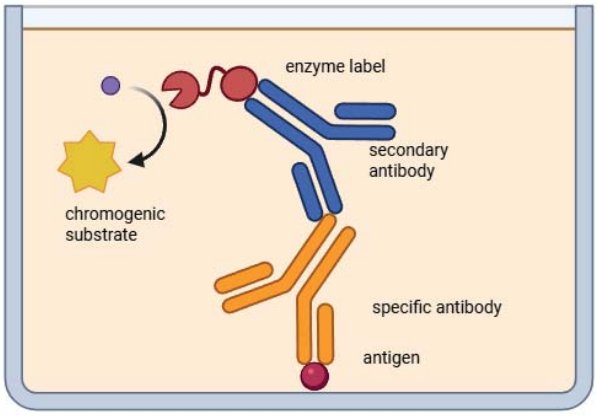

Fig. 2. **Illustration of the indirect (small i) ELISA principle (small p) [35]**

Immunological methods are based on the specific reaction between an antigen (the immunogenic structure of the pathogen) and an antibody produced by the immune system in response to it. In the enzyme-linked immunosorbent assay (ELISA), the visualization of this reaction is possible due to the introduction of an enzyme label into the system, which catalyzes the conversion of a substrate, resulting in a color change. In the solid-phase indirect ELISA, the detection of antibodies specific to the pathogen is carried out by their reaction with an antigen immobilized on a plastic support. Detection occurs via secondary anti-species antibodies conjugated with an enzyme—most often horseradish peroxidase (Fig. 2).

Commercially available ELISA test systems enable the detection of various classes of antibodies specific to *M. hominis* in blood serum, including IgG, IgA, and IgM. Serological diagnostic methods are a valuable tool in the treatment process or follow-up examination. However, they can yield false-negative results, as antibodies are often absent in individuals with a confirmed diagnosis [49]. The primary challenge in creating such tests is identifying conservative regions of the pathogen and achieving both sufficient sensitivity and specificity. Strategies that help circumvent these issues include the use of a mixture of proteins or a chimeric recombinant protein [18]. Test kits of this type, both foreign and Ukrainian-made, are present on the market.

Three test kits from the manufacturer DRG International Inc. (USA) allow for the detection of IgG, IgA, and IgM antibodies. However, only one of them, "*Mycoplasma hominis* IgG ELISA," is intended for in vitro diagnostics, while the others are solely for research purposes. These test kits are designed for the semi-quantitative determination of antibodies specific to the P120 surface protein. A unique feature of the Mycoplasma IgA and Mycoplasma IgM test kits from

this manufacturer is the pre-incubation of samples with a sorbent containing antibodies against human immunoglobulin class G, which prevents competitive inhibition by this class of antibodies [50]. The assortment of test kits manufactured by LLC "Vitrotest Bioreagent" (Ukraine) also includes three kits for detecting different antibody classes (G, M, and A) in blood serum or plasma [51].

Regarding ELISA test systems for detecting *M. hominis* antigens, information on such commercial kits is currently unavailable. The reason for this is the challenges associated with the intracellular localization of the microorganism and the low content of these antigens in clinical samples. Similar attempts to detect *M. pneumoniae* have sought to implement antigen detection in a sandwich ELISA format, utilizing both polyclonal and monoclonal antibodies against its surface lipoproteins. Although such a system provided good sensitivity — 20 ng/ml — the method did not see further development [52]. Despite the absence of diagnostic test systems built on the sandwich ELISA principle for detecting *M. hominis*, attempts to create the basic components — antibody pairs — have already been made. The American company Creative Diagnostics offers two clones of monoclonal antibodies (C676N and C675N) that are reportedly intended for use in sandwich ELISA. Conversely, the detection of mycoplasmas in fixed preparations using labeled antibodies is the closest method in principle to antigen detection in samples.

Immunocytochemical (ICC) and immunohistochemical (IHC) methods are microscopic detection techniques that rely on the specific antigen-antibody reaction. However, in this case, the antigen being detected — specifically, the surface antigens of the pathogen's cells — is present in the examined sample. This method is used for analyzing microscopic tissue preparations from the urogenital tract. These techniques emerged with the advent of fluorescent labeling and enable the detection of pathogen antigens in a fixed sample using labeled, specific monoclonal or polyclonal antibodies.

For a long time, antibodies with fluorescent tags were the predominant choice. However, this presented particular difficulties, such as the natural autofluorescence of some tissues, as well as the need for specialized equipment. Currently, chemical labeling methods are also widely used, including labeling with peroxidase or alkaline phosphatase, as well as the use of biotin-avidin-peroxidase or peroxidase-antiperoxidase systems. Additionally, there are other limitations: during the fixation process, antigen epitopes are often modified or destroyed due to changes in medium polarity or fixative temperature. However, efforts are made to minimize these drawbacks by reducing the fixation time to a minimum [53].

Reagents for detecting *M. hominis* antigens in fixed samples are typically monoclonal antibodies. For most reagents currently available on the market, the target is P120; that is, immunizing mice with antibody clones against this surface antigen (e.g., products from LSBio, USA, and GeneTex, Inc., USA). However, the majority of reagents from these and other manufacturers are not diagnostic systems and are intended solely for research purposes, including Western blot and immunofluorescence assays, as well as immunohistochemistry.

Immunochromatographic tests (ICTs), lateral flow assays (LFAs), or rapid tests are excellent tools for use in hospitals, known as point-of-care testing (POCT), as well as in home settings due to their simplicity and speed.

This method is based on the biochemical interaction of antigen and antibody. A lateral flow assay (LFA) consists of four parts (Fig. 3): the sample application pad (1); the conjugate pad, where labeled tags are combined with biorecognition elements (2); the reaction membrane containing the test line and control line for the antigen-antibody interaction (3); and the absorbent pad for waste (4).

To create the system, gold nanoparticles, colored latex beads, carbon nanoparticles, and enzymes are used as labels to increase sensitivity [54].

Currently, there are no ready-made solutions for detecting *M. hominis* antigens in this format. However, immunochromatographic tests for detecting *M. pneumoniae* do exist. In these cases, the targets are ribosomal proteins L7/L12, as used in the Ribotest Mycoplasma diagnostic system (Asahi Kasei Pharma Corp., Japan), or the P1 surface protein [55, 56]. A detailed comparative analysis of these tests indicates high specificity

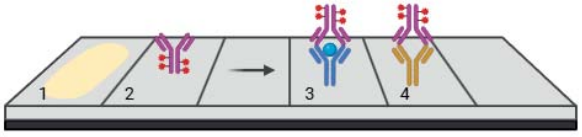

Fig. 3. **Illustration of the lateral flow assay (LFA) principle [35]**

but somewhat lower sensitivity compared to PCR [57].

New diagnostic methods have potential, but their implementation into routine practice requires time. For instance, matrix-assisted laser desorption/ionization-time of flight mass spectrometry (MALDI-TOF MS) has recently emerged as a promising tool for microorganism identification and diagnosis. The essence of the method lies in obtaining a mass profile of proteins from cellular extracts and comparing it with a spectral database; therefore, the method's limitation is the actual completeness of this database and the presence of peptide mass fingerprints of typical strains within it. The advantages of this method include high throughput, speed, and low analysis cost. Full automation is also a significant benefit: among the most renowned manufacturers of MALDI spectrometers are Shimadzu Corporation, Japan; Bruker Daltonics, USA; and JEOL Ltd., Japan, whose instruments enable the identification of mycoplasmas, as well as fungi and other bacteria [58].

Despite the high selectivity of this method for identifying specific microorganisms, especially in culture, there are significant practical obstacles. For instance, *M. hominis* was identified with 99.9% accuracy in a patient with a brain abscess; however, in this case, the culture was first isolated by the cultural method on agar medium, rather than being directly detected from a liquid sample [59]. There is still no adequate algorithm for analyzing mixtures of microorganisms, especially if some of them are non-culturable and are therefore absent from databases. One approach is to obtain the proteomic fingerprint of the sample and compare it with the expected profile of a mixture of microorganisms. Alternatively, the mixture is treated as a large metagenome and analyzed separately using powerful bioinformatics algorithms. The fact that such a summation of component spectra is not linear due to competition among proteins for desorption and ionization adds complexity to the analysis. Furthermore, the actual protein expression may differ from the model. Thus, the successful implementation of this method into routine clinical practice still requires addressing several issues [60].

There have also been attempts to develop a CRISPR-based screening test; however, this method is not yet widespread in clinical practice [61]. The known technology SHERLOCK (OraSure Technologies, USA) still faces specific difficulties that need to be resolved, primarily in terms of accuracy, as non-specific reactions lead to false-positive results. Additionally, there is a need for an extra amplification stage and specific reagent storage conditions, particularly for the guide DNA [62].

Unlike the methods above, the MycoAlert (Lonza, USA) assay is not a novel method. It is a biochemical test based on the enzymatic catalysis of ADP conversion to ATP by specific enzymes present in the mycoplasma cell. The increase in ATP can be detected using a bioluminescent reaction involving luciferase, and the released energy in the form of light is measured by a luminometer. However, such tests are not used in clinical practice and are only used for the detection of Mycoplasma contamination in cell cultures [63].

Conclusions

The complexity of detecting *M. hominis* in the diagnosis of urogenital mycoplasmosis stems from the pathogen's morphological and genetic features, its diverse immune evasion mechanisms, its regular presence in the human body, and its coexistence with other pathogens.

The widespread use of PCR methods for mycoplasma detection is based on its undoubted advantages, such as high accuracy and specificity. However, it requires specialized laboratories and equipment (which poses a problem in certain situations), as well as time, and can sometimes still yield inaccurate results. Accurate analysis can be hindered by the presence of other microorganisms in the samples, and false-negative results may occur due to the presence of inhibitors (such as hemoglobin) or as a result of DNA degradation [64, 65]. Undoubtedly, the PCR method is the preferred method for diagnosing extragenital *M. hominis* infections. Still, there is no definitive answer as to whether the hypersensitivity of PCR is beneficial for diagnosing urogenital infections [66]. Both PCR and ELISA may also be non-indicative due to the commensal nature of *M. hominis*.

One way to avoid the misinterpretation of results is to introduce a cutoff value for bacterial load, which enables the differentiation of *M. hominis* colonization in healthy individuals. On the other hand, strains that cause disease are functionally designed for adhesion rather than proliferation, thus casting doubt on the relationship between colonization level and pathogenicity [67]. Furthermore, the pervasive presence of this microorganism in healthy people is associated with high background seropositivity for IgG,

highlighting the need for confirmation of seroconversion. However, IgM antibodies may never develop, likely due to reinfections.

The relevance of ELISA-based test systems for determining *M. hominis* antibodies is determined by the speed and convenience of the analysis, along with a relatively low cost and an acceptable level of accuracy. However, antibodies to mycoplasmas will only be detectable in blood samples 1–2 weeks after the onset of the disease, making pathogen detection by this method impossible before this term [68]. A potential solution in this area is the development of a complex of sensitive ELISA assays for detecting *M. hominis* antigens in urogenital samples and antibodies in blood, which would enable rapid and effective diagnosis.

Despite all the limitations of the methods, the value of each in diagnosis is undeniable, and the appropriateness of their application should be based on the totality of information regarding the limitations discussed in the review. Therefore, the most effective strategy for detecting mycoplasmas in disease diagnosis is the simultaneous determination of their antigens in urogenital samples and antibodies in blood, which can also serve as a benchmark for improving or developing new comprehensive test systems.

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Conceptualization: T.S. Todosiichuk; Writing: L.V. Vlasenko, T.S. Todosiichuk; Review and editing: L.V. Vlasenko

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ПРОБЛЕМИ ТА СТРАТЕГІЇ В ДЕТЕКЦІЇ *Mycoplasma hominis*

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Серед захворювань, які передаються переважно статевим шляхом, одне з перших місць займає урогенітальний мікоплазмоз. Клінічні прояви захворювання, спричинені *M. hominis*, часто схожі на симптоми інших захворювань урогенітального тракту бактеріальної, вірусної та інших етіологій. Тому діагностика урогенітального мікоплазмозу, спричиненого *M. hominis* стикається з низкою проблем, незважаючи на можливість використання генетичних методів для прямого визначення ДНК цього мікроорганізму.

Мета. Комплексний аналіз особливостей *M. hominis* та сучасних методів для визначення перспективних підходів у діагностиці урогенітального мікроплазмозу, спричиненого цим мікроорганізмом.

Методика. Пошук та систематизація сучасних наукових даних та результатів щодо особливостей виявлення мікоплазм в літературі, що охоплює численні бази даних, включаючи *PubMed, Web of Science, Scopus, Google Scholar* та інші відповідні джерела.

Результати. Проведено комплексний аналіз особливостей морфології, генетики та механізмів взаємодії з організмом людини *M. hominis*. Розглянуто можливості сучасних методів виявлення такого збудника урогенітального мікоплазмозу, серед яких генетичні, імунологічні, імуноцитохімічні, імуногістохімічні методи, а також імунохроматографічні тести, тести бічного потоку або експрес-тести.

Висновки. Складність виявлення *M. hominis* у процесі діагностики урогенітального мікоплазмозу обумовлена морфологічними і генетичними особливостями збудника, різноманітними механізмами маскування від імунної системи, присутністю його в організмі людини у нормі, а також у поєднанні з іншими патогенами. Найбільш ефективною стратегією виявлення мікоплазм у діагностиці захворювань, є одночасне визначення їхніх антигенів в урогенітальних зразках та антитіл у крові, що може бути й орієнтиром для удосконалення або розроблення нових комплексних тест-систем.

Ключові слова: *M. hominis*, урогенітальний мікоплазмоз, полімеразна ланцюгова реакція, імуноензимний аналіз, антигени, антитіла, діагностика.