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Mycoplasmal PID: A Review of Natural and Experimental Infections

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The present report is a review of data assuming an etiological relationship between pelvic inflammatory disease (PID) and *Mycoplasma hominis*. Thus the organism can be isolated from the vagina/cervix more frequently in PID patients than in any other clinical group, i.e., in half to three-fourths of all such cases. One-fourth of PID patients develop a significant antibody response to *M. hominis* during the course of the disease. The antibody response can be detected by indirect hemagglutination tests. Grivet monkeys infected experimentally with *M. hominis* develop PID, predominantly parametritis; the infection seems to spread via lymphatics to the parametria. These animals develop a significant antibody response. The animals, like naturally infected women, develop a marked increase in the serum level of IgM. In tissue cell cultures of human fallopian tubes experimentally infected with *M. hominis*, a decrease of the mucociliary wave activity occurs.

So far, few clinical data support an etiological role for *Ureaplasma urealyticum* in PID. In grivet monkeys, the organism does not produce PID.

INTRODUCTION

Pelvic inflammatory disease (PID) is increasingly common. This increase seems to be the result of the pandemic situation with regard to sexually transmitted diseases (STD) that has prevailed in recent years in several developing countries. Sterility as a sequelae to PID [1] has reached such proportions that it has even begun to influence the population structure.

The etiology of PID is multifactorial. Approximately 15 percent of PID cases in western industrial countries are associated with iatrogenic measures, such as insertion of an intrauterine contraceptive device (IUD) [2]. Most of the remaining cases seem to be "venereal" in origin, that is, occurring as an STD [3]. Also among "iatrogenic" PID cases, however, such agents can be the cause of PID, as they often occur in asymptomatic carriers, including women consulting for birth control.

Chlamydia trachomatis and *Neisseria gonorrhoeae* are among the most common causative agents of PID. Anaerobic bacteria seem to be more often associated with PID in some geographic areas than in others (cf. [4]). *Mycoplasma hominis*, or pleuropneumonia-like organisms (PPLO), have also been considered an etiological agent of PID [4,5,6,7,8,9,10,11,12,13,14,15,16,17], though the proportion of such cases is, however, still not clear.

The present study discusses the current knowledge concerning PID associated

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with *M. hominis* and *Ureaplasma urealyticum*. The occurrence of these organisms in the lower female genital tract is also briefly considered.

OCCURRENCE OF *MYCOPLASMA HOMINIS* AND *UREAPLASMA UREALYTICUM* IN THE LOWER FEMALE GENITAL TRACT

Mycoplasma hominis

The first human isolate of mycoplasma was made as early as 1937 by Dienes and Edsall [18] in pus from a Bartholin's gland abscess. The strain seems to have been a strain of *M. hominis*. This species was named in 1956 by Edward and Freundt [19].

M. hominis represents the vast majority of isolates of "classic" or large colony-forming mycoplasmas from the genital tract (cf. [20]).

M. hominis has been found in 40–70 percent of all women with lower genital tract infection (LGTI). In women lacking signs of LGTI, significantly lower percentages of carriers of *M. hominis* have been reported. The occurrence of *M. hominis* in healthy controls varies considerably between different studies. In general it would seem that 5–20 percent of all sexually active women of child-bearing age seem to harbor *M. hominis*. After the menopause, the incidence decreases. The proportion of *M. hominis* culture-positive women is correlated to sexual experience, i.e., the carrier rate increases with the number of sexual partners a person has experienced [21].

In the vast majority of women colonized by *M. hominis,* the vaginal smear has a characteristic "dirty" appearance. Correlation has also been found between slight to moderately severe dysplasia of cervical epithelial cell and colonization with *M. hominis*. Antibiotic treatment resulting in the eradication of *M. hominis* was followed by disappearance of the "dirty" appearance of vaginal smears [22].

The association of *M. hominis* with cervicitis and vaginitis is still ill-defined.

M. hominis can occur also in the upper respiratory tract, i.e., in 3–4 percent. In persons practicing fellatio, the corresponding figure was up to almost 15 percent. In experimental studies, *M. hominis* produced exudative pharyngitis [23]; however, whether this is also the case in naturally acquired infection is not known.

Ureaplasma urealyticum

Ureaplasmas occur in the lower genital tract of the majority of women of child-bearing age [24]. As with *M. hominis,* the incidence of *U. urealyticum* is influenced by age, sexual activity, and hormonal status; the highest frequency is demonstrated in pregnant women [21,24]. No significant difference in the occurrence of *U. urealyticum* between women with and without signs of genital infection has been demonstrated [24].

ASCENDANT INFECTIONS IN WOMEN WITH *MYCOPLASMA HOMINIS*

Isolation Studies

Both *M. hominis* and *U. urealyticum* can be recovered form the fallopian tubes and the cul-de-sac.

A number of authors (Dienes et al. [5], Randall et al. [16], Hirsch [7], Gotthardson and Melén [6], and Stokes [17]) reported the recovery of PPLO in women with PID (in some instances later identified as *M. hominis*) from the fallopian tubes or tubovarian abscesses. The strain isolated by Freundt [25] represented *M. fermentans* (which is a rare finding in the genital tract).

In a series of 50 women with laparoscopic signs of acute salpingitis, Mårdh and Weström [11] found four (8 percent) to harbor *M. hominis* in pure culture in the

TABLE 1
Isolation of *Mycoplasma hominis* from Women with Acute Salpingitis

Author, Publication Year [Reference]	No. (%) Culture-Positive/No. of Women Studied		
	Cervix	Fallopian Tubes	Cul-de-sac
Mårdh and Weström, 1970 [11]	31(62)/50	4(8)/50	0(0)/50
Eschenbach et al., 1975 [27]	14(72)/204	NS	2(4)/54
Sweet et al., 1980 [26]	11/24	0(0)/24	1(4)/24
Møller et al., 1981 [15]	91(55)/166	NS	

NS = not studied

tubes. The organism was isolated from the cervix in 31 (9 percent) of the women. None of 34 women without laparoscopic signs of salpingitis was found to have *M. hominis.* Laparoscopy, in addition to permitting sampling from the oviduct, provides an objective diagnosis of PID [3].

In another series, Sweet and co-workers [26] found one of 26 PID patients to be culture-positive for *M. hominis* from cul-de-sac. Eschenbach and co-workers [27] isolated *M. hominis* from the cervix of 145 of 204 women with PID. From 54 of these 204 women samples were aspirated from cul-de-sac. *M. hominis* was recovered from two of the 54 samples [15] of 166 Danish PID patients, 91 (55 percent) harbored *M. hominis* in the cervix. In another Danish study [10], 30 of 95 PID cases had positive culture for *M. hominis* from the cervix. This latter study also showed that double infections with *M. hominis* and other STD agents are common. The results of some culture studies in PID patients are summarized in Table 1.

Serological Studies

Several methods have been devised to detect antibodies to *M. hominis.* They include complement fixation [9,14], metabolic inhibition [28], growth inhibition [29], and indirect hemagglutination (IHA) tests [30,31]. IHA tests—performed as described by Krogsgaard-Jensen [30]—have been found to be superior to the other tests mentioned for detection of antibodies to *M. hominis* in women. A similar experience has been reported in grivet monkeys, experimentally infected with *M. hominis,* which developed PID [32].

Antibodies to *M. hominis* as demonstrated by CF tests occurred in nine (33 percent) of 27 women with PID [21], and in 20 (39 percent) of 51 women with PID [9]. IHA antibodies to the organism occurred in 28 (54 percent) of 52 women in whom the diagnosis of PID was verified by laparoscopy, as compared to 11 percent in 56 women in whom laparoscopy showed no infectious conditions, e.g., cysts and endometriosis [12]. Eschenbach et al. [27] found that 17 (22 percent) of 84 PID patients had antibodies to *M. hominis.* Of 60 consecutive cases with acute salpingitis, IHA antibodies to *M. hominis* occurred in 24, ten of whom developed a significant titer change [13]. Antibodies to *C. trachomatis* and *N. gonorrhoeae* occurred in 80 percent and 11 percent, respectively, of these 60 PID patients. A significant antibody response to these two latter organisms occurred in 20 and three women, respectively [13]. In a series of 166 Danish women with PID, 23 percent showed a significant antibody response to *M. hominis.* Interestingly, 14.6 percent of the 75 *M. hominis* culture-negative cases also developed such an antibody response [15] (Table 2).

Increased serum levels of IgM (> 155 mg per 100 ml) was demonstrated in 18 of 53 women with salpingitis and serological evidence of a significant antibody

TABLE 2
Antibody Response to *Mycoplasma hominis* in Women with Acute PID from Whom Consecutive Sera Were Obtained

Author, Publication Year [Reference]	Type of Antibody Test Used	No. of Women Tested	Antibodies Found *n* (%)	Significant Body Response *n* (%)
Melén and Gotthardson, 1955 [14]	CF	27	9(31)	4(15)
Lemcke and Csonka, 1962 [9][a]	CF	11	11(100)	2(18)
Mårdh and Weström, 1970 [12]	IHA	29	16(55)	9(31)
Eschenbach et al., 1975 [27]	MC	77	?	17(22)
Møller et al., 1981 [15]	IHA	166	70(42)	50(30)
Mårdh et al., 1981 [13]	IHA	60	24(40)	10(17)

[a]All together 51 salpingitis patients were tested, using three different antigens which detect antibodies to *M. hominis* in 28, 26, and 25 cases, respectively. From 11 patients, all of whom had CF antibodies to the organism, consecutive serum samples were obtained.

CF = complement fixation; IHA = indirect hemagglutination; MC = mycoplasmacidal antibody test

response to *M. hominis* [33]. There was a significant ($p < 0.001$) correlation between the IgM increase and the presence of serum IHA antibodies to *M. hominis*. The IgM level of 200–299 μg per 100 ml was found in 11 percent and of > 300 in seven of 53 PID cases. The maximum level was 380 mg per 100 ml. In a few cases there was also a slight increase in the serum IgG and IgA [33].

As in *M. pneumoniae* infections [34] the increased IgM levels found in PID patients presumably could not be explained by the formation of antibodies specifically directed against *M. hominis*. Most probably they denote an unspecific stimulation of the immune system [33]. So far, there is, however, no evidence that *M. hominis* acts as a polyclonal B-cell stimulator. Antibodies to *M. pneumoniae* did not occur more commonly in the PID patients than in the general population of the study area [33]. A corresponding rise in the IgM antibody level was found in grivet monkeys experimentally infected with *M. hominis,* which developed parametritis rather than endosalpingitis [32].

Lack of IgM to IgG Conversion

Some patients with *M. hominis*-associated salpingitis did not show conversion from essentially an IgM to an IgG antibody response to *M. hominis*. Persistence of mainly IgM antibody formation was found for several months [33]. This is in conformity with findings in patients with *M. pneumoniae* [34].

Mode of Infection Spread with Mycoplasma hominis *to the Upper Genital Tract*

How *M. hominis* ascends in the female genital tract is not known. Experiments in grivet monkeys (see the section following), however, suggested spread via lymphatics and blood vessels rather than canalicularily from the uterus to the tubes [35].

EXPERIMENTAL INFECTION WITH *MYCOPLASMA HOMINIS* IN MONKEYS

In grivet monekys, parametritis and salpingitis developed within three days when *M. hominis* was inoculated directly into the fallopian tubes. Thus there was swelling and hyperemia of the tubes and the parametria became edematous. The tubal

subserosa was heavily infiltrated with lymphoid cells and an increased number of polymorphonuclear leukocytes. In the parametria many lymphocytes and some granulocytes were found, as well as fat necrosis and granulation tissue. After four to five weeks the upper genital tract of the monkeys was virtually normal again [36].

PATHOGENETIC MECHANISMS OF *MYCOPLASMA HOMINIS* IN PID

In experimentally infected explants of human fallopian tubes, the mucociliary wave activity can be registered by the light-beam reflex method [37]. Qualitative and quantitative studies of the ciliary beating can be made. The tubal specimens are placed in a chamber with regulated gas temperature and humidity, where they are lighted through a glass fiber optic. The changes in the light reflexes caused by the beating cilia are registered by a photomultiplicator. Inhibitory effects on the ciliary activity by infection can be registered by such measurements. Infection with *M. hominis* caused a reduced ciliary activity [37].

In experimentally infected explants of human tubes, *M. hominis* caused swelling of the cilia, either of the tips or the entire cilia. The mechanism underlying the swelling is not known. An arginine deficit in the tissue culture medium, caused by the metabolism of *M. hominis,* might mediate the ciliostatic effect.

ASCENDANT INFECTIONS WITH *UREAPLASMA UREALYTICUM* IN NATURALLY ACQUIRED INFECTIONS IN WOMEN AND IN EXPERIMENTAL INFECTIONS IN MONKEYS

U. urealyticum has been recovered from the fallopian tubes [27], from cul-de-sac of a woman with a tubovarian abscess [38], and in a woman with signs of acute salpingitis [11]. A significant antibody response, i.e., of metabolic-inhibition antibodies, to *U. urealyticum* (to a strain isolated from the fallopian tube of a PID patient) has been demonstrated in one woman with acute salpingitis [24]. Eschenbach et al. [27] found 15 of 204 PID patients to develop ureaplasmacidal antibodies. Sweet et al. [26] found *U. urealyticum* in cul-de-sac fluid in four of 26 women with PID (also in the fallopian tubes of one of them).

In animal experiments, viz., in grivet monkeys, no pathogenic effect of *U. urealyticum* was demonstrable [39]. Mycoplasmas have also been recovered from the bovine oviduct [40]. In experimentally infected heifers, ureaplasmas and mycoplasmas could induce damage to the oviduct mucosa [41,42].

TREATMENT OF MYCOPLASMAL PID

M. hominis is resistant to betalactam antibiotics and nitroimidazoles, which are drugs often used in the treatment of PID.

Tetracycline has an effect against *M. hominis* both *in vitro* [43] and *in vivo* [44]. Minimum inhibitory concentrations (MIC) of various tetracyclines of 0.2–0.8 mg/l have been found [43]. The corresponding values for doxycycline were 0.1–0.4 mg/l. *U. urealyticum* likewise is sensitive to tetracyclines (MIC = 0.4–1.6 mg/l).

A tetracycline-resistant strain of *M. hominis* was isolated from a pregnant woman who had arrived in Sweden from Jordan the day before sampling and delivery [45]. The MIC of tetracycline for this strain was 32 mg/l. A tetracycline-resistant strain of *M. hominis* could also be isolated from the cerebrospinal fluid of her offspring, who had signs of meningoencephalitis [45]. Strains of *U. urealyticum* with a decreased susceptibility to tetracycline have also been reported to be relatively common.

M. hominis generally can be eradicated from the female genital tract by a standard course of tetracycline for one or two weeks [44]. This is in contrast to *U.*

urealyticum, which often persists after standard courses with tetracycline. Thus the organism has been found in up to one-third of the patients given such therapy [44].

In contrast to *M. hominis, U. urealyticum* is susceptible to erythromycin (MIC = 0.2–0.8 mg/l) [43].

In the case of deep, complicated genital infections and in systemic infections by *M. hominis,* at least two weeks of tetracycline treatment should be given.

The risk that *M. hominis* can spread to the newborn infant, resulting in infections of the skin [46,47], the eyes [48], and the central nervous system [45], must not be disregarded.

SEQUELAE OF MYCOPLASMAL PID

No studies have so far been presented concerning sequelae of PID stratified with regard to the major common etiological agents of PID, including *M. hominis.* The risk of sterility after PID is known to be high—roughly 10–15 percent after one episode of the disease, 35 percent after two, and 75 percent after three or more such episodes [1]. Chronic abdominal pain and increased risk for etopic pregnancy [49] are other common sequelae after PID. Whether such sequelae also occur after *M. hominis*-associated PID is not known.

CHALLENGES FOR FUTURE RESEARCH

Further studies on antigenic differences [50] among organisms now classified as *M. hominis* ought to be performed.

More epidemiological data concerning *M. hominis* infections of the upper genital tract are required. So far, most epidemiological reports have derived from the Scandinavian countries or from the U.S.

Whether there is a discrepancy between findings in grivet monkeys experimentally infected with *M. hominis* and naturally infected human beings with PID remains to be determined. In the monkeys, the parametria rather than the fallopian tubes seem to be affected.

Clinical observations indicate also that the parametria might be involved in PID in women who are culture-positive for *M. hominis* and in whom a significant antibody response to the organism can be demonstrated. Furthermore, a biopsy of the parametria of such a woman revealed similar inflammatory changes as those seen in monkeys experimentally infected by *M. hominis* [Møller BR: personal communication]. Further clinical studies on parametritis are required.

More studies are also needed to establish pathogenetic mechanisms in *M. hominis* infections, including PID.

Finally, possible sequelae of mycoplasmal PID must be further studied.

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