

A Review of *Pasteurella pneumotropica*

Pasteurella pneumotropica is an opportunistic organism prevalent in many commercial and research colonies of rodents. The incidence of clinical disease associated with this organism is low, and very few research interactions have been documented in asymptomatic infection. In the presence of primary pathogens, this organism, like other opportunistic organisms, potentiates the severity of disease. Many disease reports attributed to *P. pneumotropica* can be explained by the presence of concurrent infections with other organisms or the failure to control environmental variables.

Complete isolation, identification and characterization of this organism from clinical isolates is not as simple or reliable as might be assumed. There is no well-established correlation between its specific biotypes and their pathogenic potential.

The broad host range, which may include man, makes exclusion from research colonies difficult without rigorous containment procedures and the elimination of human/animal contact. In the view of Charles River Laboratories, the presence of *P. pneumotropica* should be noted but not regarded as cause for destruction of production colonies or classification of animals as inappropriate for research.

AGENT DESCRIPTION

P. pneumotropica belongs to the family Pasteurellaceae, which also includes the genera *Haemophilus* and *Actinobacillus*. *P. pneumotropica* is a gram-negative short rod or coccobacillus (0.5 x 1.2µm). On primary culture the organism grows well on blood agar, producing by 24 hours convex (0.5-1.5mm) light gray to yellow colonies. It shows bipolar staining and produces acid but not gas from dextrose, glycerol, inositol, lactose, maltose and mannose. It is also oxidase-, urease-, and catalase-positive and can reduce nitrate to nitrite.

P. pneumotropica can be divided into several biotypes based on the reactivity of various isolates on differential media (Heyl, 1963; Hooper and Sebsteny, 1973; Simmons and Simpson, 1977). Heyl proposed two such groups based on xylose and inositol reactivity. Other studies suggest that even more biotypes may exist. Biotype classification systems based on cell-wall lipids, structural proteins, or DNA analysis have been proposed and may also prove useful. Unfortunately, correlations between specific biotypes and pathogenicity have not been firmly established, nor have specific criteria been developed for evaluating pathogenicity.

HOSTS

P. pneumotropica has been isolated from mice, rats, hamsters, guinea pigs, rabbits, cats, and numerous other laboratory animals (Erfle and Neuman, 1980; Kunstyr and Hartman, 1983; Sparrow, 1976; Wheeler, 1967). Surveys of conventional (i.e., nonaxenic or nonassociated) rodent colonies have revealed a very high incidence of asymptomatic infection with this organism (Sparrow, 1976; Saito et al., 1977). In most cases it can be recovered from the respiratory tract, the urogenital tract or conjunctiva.

Whether humans serve as an important host of *P. pneumotropica* is unclear. Isolations from humans were reported by Henriksen in 1962. Weaver et al. (1985) reported on nine separate isolates of *P. pneumotropica* of the Jawetz or Heyl biotypes from humans. While not specified in the report, it may be inferred that these isolates were associated with human disease, including bite wounds from animals. (No information is available to show whether a latent carrier state exists in humans as exists in animals.) In this same review, a distinctively separate biotype originally described by Henriksen was reclassified as a separate *Pasteurella* species (Frederikson, 1981). It has been isolated from humans having infections associated with animal contact.

Clearly, the classification of *P. pneumotropica* and its various biotypes has evolved significantly from its initial description and isolation (Jawetz, 1948). Precise definition of its host range and other biological parameters is very difficult, particularly as its biochemical profile is very similar to other species within the genus and to other members of the Pasteurellaceae (Van der Shaaf et al., 1970). Several members of the genus *Actinobacillus* may easily be confused with *P. pneumotropica* unless the isolates are carefully characterized (Lentsch and Wagner, 1980).

BASIC BIOLOGY

P. pneumotropica is most likely transmitted by direct contact or fomites. According to Jawetz and Baker (1950), it has an affinity for the respiratory tract, as experimental parenteral or intranasal inoculation will yield latent infection only in that area. The organism does not appear to be vertically transmitted and is not secreted in the milk. Moore et al. (1973) reported its isolation from the feces of "gnotobiotic" rats, which suggests vertical transmission. However, other studies offer indirect evidence that "vertical transmission" during rederivation may result from isolator contamination or uterine infection with the organism (Hoag et al., 1962; Brennan et al., 1965; Ward et al., 1978). The organism can certainly be isolated from the urogenital tract and could conceivably lead to uterine infection and potential contamination of the developing fetus.

Studies by Blackmore and Casillo (1972) have established that *Pasteurella* infection of the urogenital tract is a result of ascending vaginal infection, probably from a respiratory source. As most laboratory rodents are coprophagic and often groom the genital area, oral and nasal secretions can easily reach the genital orifices. Blackmore and Casillo found uterine infections to be short-lived. This finding, coupled with the lack of evidence for transplacental passage of the organism, argue against true vertical transmission.

As noted above, the organism has been isolated from a high percentage of apparently healthy animals in many colonies. It may be isolated from numerous organs (Kunstyr and Hartman, 1983), but the conjunctiva, skin, lungs, and other areas of the respiratory tract are by far the most common sites, regardless of the presence or absence of disease.

CLINICAL DISEASE AND PATHOLOGY

For the most part, *P. pneumotropica* is an opportunistic organism seldom associated with clinical disease. Jawetz' initial description was soon followed by more detailed studies (Jawetz, 1950; Jawetz and Baker, 1950) using experimentally induced disease to study its pathogenesis. In these studies, extensive serial passages in mice (up to 16 passages) and maintenance of the organism on artificial media for no more than eight hours were necessary to produce respiratory disease. Moreover, relatively large intranasal inocula were required. Jawetz noted that *P. pneumotropica* was not responsible for spontaneous outbreaks of respiratory disease in mice. This observation holds true today for mice and other laboratory rodents.

Early studies of respiratory disease in rodents using *P. pneumotropica* were often hampered by a lack of well-defined animals free of latent or concurrent infections with other organisms. For example, Jawetz stated that the mice used in his studies had a natural *Chlamydia trachomatis* infection and "spontaneous pulmonary consolidation" possibly related to Sendai virus, *Mycoplasma pulmonis* or other agents. Burek et al. (1972) have reinforced the point that *P. pneumotropica* is not a primary respiratory pathogen in rats. In their study, despite frequent isolation of the organism from experimentally infected axenic and conventional rats, no lung lesions or other respiratory tract lesions could be produced. Thus, despite its apparent predilection for the respiratory tract, it does not appear to produce respiratory disease.

Systemic infection with *P. pneumotropica* has not generally been reported, nor have pathologically demonstrable changes associated with septicemic dissemination to internal organs. Uterine infections have been noted (Ward et al., 1978; Brennan et al., 1965; Blackmore and Casillo, 1972; Hoag et al., 1962) but without good correlation between the presence of *P. pneumotropica* and demonstrable histological changes. Moreover, predisposing factors such as concurrent infections, age or stress were not ruled out in these reports.

As stated previously, *P. pneumotropica* is a common inhabitant of the urogenital tract. Its presence in the uterus is associated mainly with ascending vaginal infections of short duration caused by grooming or coprophagy. In the male, bulbourethral gland infection was reported by Sebesteny (1973), but its incidence was not discussed, nor were other possible contributing factors.

Subcutaneous and cervical abscesses have been reported in both rats and mice (Moore and Aldred, 1978; Weisbroth et al., 1969; Wilson, 1976; Van der Shaaf et al., 1970) but in general, the incidence of cutaneous abscesses was quite low or not defined in these reports. Moreover, predisposing conditions such as cleanliness of the environment, mechanical abrasions and concurrent infection with other organisms were not defined.

The presence of *P. pneumotropica* on cutaneous surfaces or in the environment has not been studied. Limited experimental evidence (Van der Schaaf et al., 1970; Sebesteny, 1973) indicates that the organism can cause cutaneous abscesses when injected subcutaneously in pure culture in mice and rats. Unfortunately, many opportunistic organisms will do so when injected in pure culture, so this evidence does not establish *P. pneumotropica* as a primary cutaneous pathogen in rodents.

Of lesions associated with *P. pneumotropica*, those of the eye and ocular adnexa are probably the most frequently documented. In mice, conjunctivitis (Needham and Cooper, 1975; Wagner et al., 1969), panophthalmitis (Weisbroth et al., 1969), and dacryoadenitis (Needham and Cooper, 1975; Wagner et al., 1969) have been reported. Unfortunately, reports of ocular and ocular adnexa involvement fail to eliminate other micro-organisms and physical factors as potential causes. For example, reports on rats by Young and Hill (1974), Hill (1974), Moore (1979), and Roberts and Gregory (1980) failed to eliminate Sialodacryoadenitis virus (SDAV) as a potential cause of the conjunctivitis noted. SDAV is a relatively common and highly infectious viral disease of laboratory rats. It causes extensive temporary destruction of the lacrimal glands, which may result in various ocular signs compatible with the lesions noted in many of these reports.

In some studies, other potential infectious co-pathogens were isolated from the ocular lesions, e.g., *Mycoplasma* sp. and *Streptobacillus moniliformis* (Young and Hill, 1974). This suggests a multi-factorial etiology and does not establish the pathogenicity of *P. pneumotropica*. Similarly, the report of Roberts and Gregory (1980) describes concurrent infection with *M. pulmonis* and does not contain a totally convincing biochemical identification of *Pasteurella* (in this case, *P. multocida*). None of these reports explores the possibility that abrasions from fine particles of bedding or feed may be primary inciting factors, with *P. pneumotropica* serving only as an opportunist.

In mice, the incidence of ocular lesions associated with *P. pneumotropica* appears to be quite low. Needham and Cooper (1975) reported an incidence of 45 affected animals out of 72,000 used over one year's time. Similarly, Weisbroth et al. (1969) reported a 9 percent incidence of *P. pneumotropica* associated lesions in animals culled for all reasons from a breeding colony. Unfortunately, they did not report the size of the colony and, hence, the true incidence of lesions. Confounding variables such as concurrent infections or environmentally associated problems were not considered.

The report by Wagner et al. (1969) does not eliminate confounding variables. Moreover, the authors failed to isolate *P. pneumotropica* from many of the infected mice and did not consider conclusive their own identification of *P. pneumotropica*. (Multiple biotypes may well have contributed to the variable biochemical profiles described in this study.) Reporting overall incidence of 3 to 4 percent, Wagner et al. (1969) concluded that changing from a shavings/sawdust bedding to dehydrated alfalfa contact bedding eliminated the potential for mechanical abrasions of the conjunctiva. Unfortunately, they offered no measurement of bedding fines or evidence regarding mechanical abrasions of the cornea and conjunctivae with and without bedding fines of various types. Plant fibers released from pelleted alfalfa are as likely to cause mechanical irritation as fines from wood products.

In general, the peer-reviewed literature fails to implicate *P. pneumotropica* as a primary pathogen under natural conditions for any of the organ systems reviewed. In studies where histopathology was conducted, lesions attributed to *P. pneumotropica* have been characterized as suppurative inflammatory processes with no particularly distinguishing features.

SYNERGISM

A number of reports suggest that this agent plays a synergistic role with viruses, mycoplasma or other bacteria in the production of disease in rodents. In rats, *P. pneumotropica* has been associated with Kilham Rat Virus (Carthew and Gannon, 1981), Sendai virus (Carthew and Aldred, 1988) and species of *Staphylococcus*, *Corynebacterium*, or *Mycoplasma* (Eamens, 1984). The studies by Carthew and Aldred and Eamens were conducted using clinical isolates collected under uncontrolled conditions. Carthew and Aldred focused on embryonic death in pregnant rats but do not mention the exclusion of KRV. Under the conditions of their study, KRV could easily have accounted for the histologic changes seen in a KRV-naive population undergoing epizootic infection with KRV. Changes noted in the lungs are very characteristic of Sendai, and there is little evidence beyond culture results to suggest any involvement of *P. pneumotropica*. Certainly, from this study, the influence of *P. pneumotropica* cannot easily be separated from the influence of other concurrent viral infections.

The report by Carthew and Gannon does not provide convincing evidence of a synergistic effect of *P. pneumotropica* with KRV. The histopathologic changes do not appear to correspond well with experimentally induced *P. pneumotropica* infection as described by Jawetz and Baker (1950). Other bacterial infections were not eliminated by culturing methods, and it was not clear what rat viruses were tested for in the animals studied. Changes were obvious in the lungs of rats simultaneously infected with KRV and *P. pneumotropica*, but the associative relationship between these two organisms has not been established.

The report by Eamens (1984) does not present a direct cause and effect relationship between *P. pneumotropica* and otitis media in the laboratory rat. Moreover, the authors suggest that there may be some confusion between classifications of various *Pasteurella* species (including *P. pneumotropica*) in their study. In addition, the concurrent effect of *P. pneumotropica* with other organisms was difficult to determine. *P. pneumotropica* can certainly be present in cases of otitis media, but its synergism with other organisms is clouded by its normal presence in the respiratory tract, which is connected to the middle ear by the Eustachian tube.

Synergistic effects in mice have been suggested between *P. pneumotropica* and Sendai virus (Jakab, 1974 and 1981) and *M. pulmonis* (Brennan et al., 1969; Saito et al., 1978). The Jakab studies demonstrate that concurrent Sendai virus infection is needed to get the additive effect of latent *P. pneumotropica* on lung lesions. While this work did not rule out other viral and bacterial infections, it showed a reasonable correlation between the combined presence of Sendai and *P. pneumotropica* and the extent of lung lesions, compared with their extent when only a single agent was present. It provides reasonable evidence that *P. pneumotropica* does have the capability of acting as an opportunist in the presence of concurrent Sendai virus infection. However, its relationship with *M. pulmonis* or other bacterial agents is less well established. Brennan et al. (1969), while unable to induce pneumonia in conventional mice infected with *P. pneumotropica*, were able to show synergism in the extent and severity of lesions when it was combined with *M. pulmonis* in both conventional and axenic mice.

Saito et al. (1978) approached the same conclusions of synergism through epidemiologic methods. They surveyed 3,270 mice from 22 breeding colonies as to the presence of *P. pneumotropica*, *M. pulmonis*, *Corynebacterium kutscheri* and Sendai, showing correlation between the presence of these organisms and the presence of lung lesions. While not as convincing as the work of Brennan et al. (1969), this study provides additional substantiation for synergistic effect.

In general, synergism as a result of secondary infection with *P. pneumotropica* in the presence of primary viral, mycoplasmal or bacterial pathogens is probably a valid phenomenon in rodents. However, other common opportunistic bacteria in the presence of those agents are likely to have similar synergistic actions. The fact remains that in the absence of a primary pathogen such as Sendai virus, this synergistic effect will not be expressed. Moreover, the magnitude of the contribution of *P. pneumotropica* in such synergism has never been established.

RESEARCH EFFECTS

Very little has been reported in the peer-reviewed literature to establish any effects of latent *P. pneumotropica* on research. The only notable exception is a report by Royston et al. (1983) showing that *P. pneumotropica* causes some alteration of alveolar-capillary barrier permeability in the respiratory tract. This study compared rats from two colonies, one with *P. pneumotropica* and one without. Other than rat viruses, the study animals were screened only for *P. pneumotropica* and *M. pulmonis*. It was assumed but not demonstrated that other bacterial populations in the respiratory tract were the same between the two groups. Alteration of the capillary permeability barrier was demonstrated between the two groups, suggesting that *P. pneumotropica* may be responsible. Unfortunately, no histologic evaluation was performed to verify the integrity of the respiratory epithelium. It can be inferred that the action of *P. pneumotropica*, if indeed it was solely responsible for these changes, occurred at the subcellular level.

Other research effects have not been demonstrated, but if *P. pneumotropica* causes ocular or ocular adnexal lesions or the formation of cutaneous abscesses, such changes could be debilitating and to some degree affect research protocols. In most clinical reports, however, these changes have been self limiting and occurred in a very small portion of the population, so their impact on research is difficult to assess.

DETECTION/DIAGNOSIS

All efforts to detect *P. pneumotropica* must discriminate between *P. pneumotropica* infection and *P. pneumotropica* induced disease. Since rodents in many colonies are asymptotically infected with this agent (in their respiratory tract, conjunctivae or other sites) without demonstrable disease, its diagnosis as a primary pathogenic agent must necessarily be one of exclusion. It is, therefore, important to characterize the bacteriologic, mycoplasmal and viral status of animals in which this diagnosis is considered, to rule out other possible causative agents and disease processes.

Considerable similarity exists between species of *Pasteurella*, *Haemophilus* and *Actinobacillus*, and at least three biotypes of *P. pneumotropica* have many similar reactions on differential media. To avoid confusion, extensive biochemical categorization of each isolate is imperative (Lentsch and Wagner, 1980; Simpson and Simmons, 1980; Hooper and Sebesty, 1974; Kunstyr and Hartman, 1983; Carter, 1984; Ackerman and Fox, 1981).

The methods used to collect primary culture material are extremely important in assuring adequate recovery of *P. pneumotropica*, especially from the respiratory tract. The success of recovery is dependent upon various factors including the age of the animal, the state of nutrition, the type and concentration of other flora at the culture site. Generally, using a nasopharyngeal wash in young animals will be more effective in detecting *P. pneumotropica* than using swabbing techniques or older animals. Various primary isolation media can be used, although blood agar plates are a common choice. An enzyme-linked immunosorbent assay (ELISA) for the detection of antibodies to *P. pneumotropica* in mice has been described (Wullenweber-Schmidt et al., 1988) and for *P. multocida* (Lukas et al., 1984; Manning et al., 1987). While these assays hold promise, a significant potential for cross reaction exists due to the large number of antigenic sites possessed by most bacteria. For this reason, such assays may have application in screening programs, but definitive diagnosis should be made by culture.

CONTROL/ELIMINATION

Because this agent is a widely distributed opportunistic organism, control of important primary pathogens and other factors that compromise host defenses are probably more important than efforts to control *P. pneumotropica*. Its total exclusion from research colonies and commercial breeders is a difficult (perhaps impossible) task, as evidenced by its high incidence in most research facilities and commercial colonies (Flynn et al., 1965; Saito et al., 1978; Kunstyr and Hartman, 1983; Sparrow, 1976). Caesarean rederivation of animals and subsequent maintenance using true aseptic techniques and gnotobiotic technology (e.g., flexible film isolators, microisolator cages) are probably necessary to exclude *P. pneumotropica* from animals used in research that may be affected by this organism. Such technology is not commonly found in most research environments and would seldom be cost-efficient, given the limited documentation of substantiated research effects of latent *P. pneumotropica* infection. The prevalence of the organism in an infected population can be greatly reduced by antibiotic therapy (Gray and Campbell, 1953; Moore and Alfred, 1978), but this approach probably has little application in most research settings.

PERSPECTIVE

As noted in the introduction, Charles River Laboratories considers that *P. pneumotropica* has negligible impact on animal health and the conduct of research. Its presence should be noted but will seldom, if ever, disqualify animals for use in the laboratory.

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