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A SEROLOGIC, FERMENTATIVE AND MORPHOLOGIC STUDY OF HUMAN, FELINE AND CANINE CULTURES OF PASTEURELLA MULTOCIDA

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A SEROLOGIC, FERMENTATIVE AND MORPHOLOGIC STUDY OF HUMAN, FELINE AND CANINE CULTURES OF *PASTEURELLA MULTOCIDA*

By

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ABSTRACT

A SEROLOGIC, FERMENTATIVE AND MORPHOLOGIC STUDY
OF HUMAN, FELINE AND CANINE CULTURES OF
PASTEURELLA MULTOCIDA

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Pasteurella multocida causes disease in many animal species. Humans become infected by contact with animals which are infected or carriers. Wound infections and infections involving various internal organs occur. Cat and dog bites are the primary cause of wound infections; an animal source cannot always be found for internal infections.

Sixteen somatic types of P. multocida have been identified by their heat stable antigen in a gel diffusion precipitin test (GDPT). Four capsular types have been identified by an indirect hemagglutination technique. Type A capsules can also be identified by a hyaluronidase test and type D capsules by an acriflavine test. Cat cultures often ferment mannitol, sorbitol and glycerol while dog cultures do not. A number of investigators have observed the colonial variation among cultures. They have found that encapsulated cultures are often iridescent.

The purpose of this study was to compare cultures of P. multocida from humans, cats and dogs according to the various criteria, viz., serologic, fermentative and morphologic. The somatic types were determined by the GDPT; the capsular types by hyaluronidase and acriflavine tests. The fermentation reactions in mannitol, sorbitol and

glycerol were determined and observations on the colonial variant were made. Information on the source of the culture and any animal association was recorded.

The following information was gained: Somatic types 1, 3, 4, 5, 8, 11 and 12 were isolated from the human cultures with types 1 and 3 predominating. Types 1, 3, and 12 were isolated from cats; types 1, 3 and 8 from dogs. Many cultures reacted with more than 1 antiserum. With these cultures, the "secondary serotype" was placed in parentheses following the "primary" serotype, e.g., 1(4). Somatic types 1(4), 4(7), 4(12) and 12(4,3) were only isolated from internal infections; types 1(7), 5(1), 8(5), 8(13) and 11 only from wounds. Type A capsules were associated with many cultures from internal infections but not cat or dog cultures. Only 2 human cultures had type D capsules. Two type D cultures were also isolated from 2 cats owned by one of the humans from whom a type D culture was isolated.

Dog, cat and human cultures of P. multocida often had different characteristics on blood and dextrose starch agars. Dog cultures were often small and dry on blood agar and non-iridescent (blue) on dextrose starch agar. Cat cultures were medium in size, creamy and blue. Human cultures were usually large, mucoid and iridescent. The fermentation pattern of these cultures was related to whether the culture was from a canine or feline source but there was not complete correlation. Many cat related cultures fermented mannitol, sorbitol and glycerol; many dog related cultures did not. However, most dog cultures which were type 3 fermented the 3 sugars. A group of cultures isolated from internal

infections fermented mannitol, sorbitol but not glycerol.

Describing cultures of P. multocida in the above ways--capsular and somatic type, fermentation pattern, colonial variant and animal association--can be useful epidemiologically. If a culture of P. multocida were isolated from a human infection and from an animal with which the human had had contact and if the culture were of the same serotype and possessed the same fermentation pattern, this information would strongly suggest that the animal was the source of the infection.

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INTRODUCTION

Pasteurella multocida is a commensal in the respiratory and gastrointestinal tracts of many animal species. It also causes diseases of great economic importance such as fowl cholera and hemorrhagic septicemia in cattle and water buffalo. Serologic studies have been done to enable vaccine formulation. Heddleston et al. have identified 16 somatic types based on a heat stable antigen visualized in a gel diffusion precipitin test.[58] Carter has identified 4 capsular types (A,B,D,E) using a hemagglutination technique.[24] Type A capsules can also be identified by their depolymerization with hyaluronidase[31] and type D capsules, by flocculation of the cells in acriflavine.[30]

Humans become infected with P. multocida by contact with animals which carry or are infected with it. Due to the high carrier rate among cats and dogs, bites by these animals are a common source of infection. Wound and internal infections occur. Carter[32] amd Smith[129] found that cat and dog cultures gave different fermentation reactions in mannitol, sorbitol and glycerol. Cat cultures usually fermented the 3 sugars; dog cultures did not.

The purpose of this study was to analyze P. multocida cultures from humans and to compare them with those from cats and dogs. Accurate records as to isolate sources were kept. The somatic type of each culture was determined by the method of Heddleston et al.[58] The capsular

type of each culture was determined by hyaluronidase[31] and acriflavine tests.[30]

Results were analyzed for the following information: serotypes associated with internal infections or wounds, differences in serotypes or fermentation patterns between cat and dog associated cultures, and putative sources for internal infections.

LITERATURE REVIEW

Systematics

Pasteurella multocida was first isolated in the late 1870's by Rivolta from an epidemic of fowl cholera[124] and by Kitt from an epidemic in wild hogs.[72] Trevisan gave this organism its first name, Bacterium cholerae-gallinarum.[141] Two other early names were B. bipolare-multocidum and B. septicaemia-hemorrhagica. The former was given by Kitt and was indicative of its staining property and its capacity to produce many diseases.[77] The latter, given by Hueppe, was descriptive of a major disease which it caused.[124] By the 1900's this organism was placed in the genus Pasteurella.[63,124] At the time, a conflict arose over whether the organism, when isolated from different animals, was the same or comprised different species.[87] Lignières was a proponent of a zoological classification. For example, a culture from chickens was named P. aviseptica; one from cattle was called P. boviseptica; one from swine was named P. suiseptica; and one from rabbits was called P. lepiseptica. This zoological system was accepted for about 30 years but during that time investigators were studying the differences among these species.[87] In 1911, Baumgarten demonstrated that the organism isolated from one animal could be pathogenic to another species.[8] Schirop, in 1908,[126] and Besemer, in 1917,[13] noted biochemical similarities among organisms isolated from different species. Specifically, Schirop found

P. vituliseptica, P. aviseptica and P. suiseptica to be similar. Mori, in 1918,[95] and Tanaka, in 1926,[138] found serologic relationships among cultures from different animal species. Mori found that antiserum to P. aviseptica agglutinated other species. Tanaka failed to find host specific serologic groups based on agglutination and complement fixation tests. In 1939, Rosenbusch and Merchant did further studies to determine the biochemical activity of these organisms. They found the species proposed under the zoological classification to be relatively homogeneous biochemically.[124] They proposed that all the different species, P. aviseptica, P. boviseptica, P. suiseptica, etc., be merged into one species to be called Pasteurella multocida, Kitt's name of 1885.[29] It first appeared as such in Bergey's Manual in 1948[11] and is currently the widely accepted name. Topley and Wilson, in 1929, suggested the name P. septica which has been used in some British Commonwealth countries.[132]

The species P. multocida is a member of the family Brucellaceae. Also included in this genus are P. haemolytica, P. pneumotropica and P. ureae. The latter is the only species of the genus that does not cause disease in animals.[132] P. gallinarum and P. aerogenes are unofficial species. The former is primarily a commensal but may cause a low-grade infection in poultry while the latter is a member of the normal intestinal flora of swine.[33] In Bergey's Manual, P. anatipestifer and P. pfaffi are placed in the category species incertae sedis. P. anatipestifer causes septicemia in ducklings. A bacterium given the name P. pfaffi was isolated from a septicemia in canaries but is now thought to have been a variant of P. multocida.[132]

Formerly, the Pasteurella genus contained, as Pasteurella species, the bacteria presently named Francisella tularensis, F. novicida, Yersinia pestis and Y. pseudotuberculosis. F. tularensis[108] and F. novicida[110] were removed from the Pasteurella genus because they differed from P. multocida in their growth requirements, their biochemical patterns, their antigenic nature and the type of disease which they produced. F. tularensis has a special requirement for cystine for growth.[38] This amino acid is provided in media such as blood cystine glucose,[117] available commercially as cystine heart agar supplemented with hemoglobin and supplement B. F. tularensis, unlike P. multocida, is a facultative intracellular parasite which elicits a characteristic granulomatous response in its host.[38]

The place of P. pestis and P. pseudotuberculosis in the taxonomic scheme was evaluated by Smith and Thal using a numerical technique.[131] Reactions in 58 biochemical tests including antibiotic susceptibilities were determined for each Pasteurella species. The Pasteurella species were then grouped in pairs and the number of test results the two had in common were determined. By dividing this number by 58, the percentage similarity between the two was determined. By this method, P. pestis and P. pseudotuberculosis were found to be 85% similar to each other but at most 65% similar to any of the other Pasteurella species. Conversely, there was found to be 75 to 85% similarity among P. multocida, P. ureae, P. haemolytica and P. pneumotropica. In addition, P. pestis and P. pseudotuberculosis are oxidase negative while the other 4 Pasteurella species are oxidase positive.[131] The differences appeared to support Van Loghem's recommendation of 1945 that P. pestis and

P. pseudotuberculosis be removed from the genus Pasteurella and be put in the genus Yersinia.[143] Sneath and Cowan confirmed these differences. From a general computer survey in 1965, they found P. pestis to be closely related to other species of Enterobacteriaceae.[133,139]

In addition to biochemical differences and different temperatures for optimum growth, the former Pasteurella species have G + C contents different than P. multocida. The G + C content of P. tularensis (based on 1 strain) was 36%; that of P. pseudotuberculosis was 43 to 48%; and that of P. pestis was 46 to 48%. The G + C content of P. multocida ranged from 37 to 43% based on twelve strains.[48]

Biochemical and Morphologic Properties

Species of the genus Pasteurella are gram negative coccobacilli, measuring 1.4 ± 0.4 by 0.4 ± 0.1 μm. Nutritionally they are chemoorganotropic, growing better in the presence of blood but not requiring it.[132] Hematin or other chemicals which reduce hydrogen peroxide, such as sodium sulfite, sodium thiolacetate, sodium pyruvate or manganese dioxide, can replace blood. These compounds are necessary for aerobic growth.[73] Minimal nutritional requirements vary[132] but Berkman found that the addition of pantothenic acid and nicotinamide was required to grow P. multocida on a basal media comprised of gelatin, salts and amino acids.[12] Concerning atmospheric requirements, the Pasteurella species are aerobic and facultatively anaerobic. The temperature range for growth is 22° to 42° C with 37° C being optimal. Metabolically, they ferment glucose and other carbohydrates producing small amounts of acid

but no gas. Other biochemical reactions which are characteristic include the following: catalase and oxidase positive, acid over acid on TSI, methyl red, Voges-Proskauer and gelatinase negative, non motile, positive for reduction of nitrate to nitrite, lysine decarboxylase and arginine dihydrolase negative and ornithine decarboxylase positive.[132]

The Pasteurella species can be differentiated on the basis of the reactions presented in Table 1, on the following page.

In the gram stained smear, P. multocida can exhibit bipolar staining or pleomorphism on initial isolation or under reduced oxygen tension. This also occurs in other gram negative bacilli. Colonies on blood agar can be large and mucoid, medium sized and smooth or small and rough.[29] By obliquely transmitted light, colonies on dextrose starch agar may demonstrate iridescent or blue optical properties or be intermediate. Capsules can be demonstrated on organisms from mucoid and iridescent colonial variants.[44]

Biotypes

Numerous studies of the biochemical properties of P. multocida have been performed. The purpose of these studies was to establish biotypes to identify strains from different host species. Khalifa was the first to correlate biotypes with immunotypes. He identified 3 immunotypes by active and passive immunization and associated them with fermentation patterns in xylose, arabinose and mannitol.[76] Rosenbusch and Merchant did similar studies. They found 3 serologic types by tube agglutination reactions and correlated them with differential fermentation of xylose, arabinose and dulcitol.[124] Roberts associated his types

Table 1. Differentiation of *Pasteurella* species.

	MacConkey	Hemolysis	Indol	Urea	Glucose	Lactose	Mannitol
P. multocida	-	-	+	-	A	N	(A)
P. haemolytica	+	β	-	-	A	(A)	A
P. ureae	-	α	-	+	A	N	A
P. pneumotropica	-	-	+	+	A	(A)	N
P. gallinarum	-	-	-	-	A	N	N
P. aerogenes	+		-	+	A (gas)	N	N
P. anatipestifer	-	-	-	-	N	N	N

A - fermentation; N - no fermentation; (A) most ferment; 29, 33, 43, 132

I and II with differential arabinose and xylose fermentation.[120] Table 2 summarizes the correlation of these 3 biotypic systems.

Table 2. Correlation of some biotypes, serotypes and immunotypes.

Serotype	Immunotype		Biotype		
Rosenbush & Merchant	Khalifa	Roberts	Arabinose	Dulcitol	Xylose
I	A	II	A	A	N
II	C	I	N	N	A
III			A	A	A

Heddleston performed biochemical tests on 1268 cultures from many animal species and tabulated them. Cultures from the following animal species varied from the other animal cultures studied in the following manner: more than 50% of sheep cultures did not ferment glycerol; waterfowl cultures usually fermented arabinose, and dog cultures generally fermented dextrin, maltose and trehalose but not mannitol or sorbitol.[62] From this same study, Heddleston found that human cultures resembled cultures from other animal species in most biochemical reactions. The exceptions were trehalose and xylose fermentation. However, cultures from most animal species were variable in these 2 sugars. From this study it was evident to Heddleston that sugar fermentation results were quite variable. As a consequence, he did not identify biotypes related to animal species from this study.[62]

Carter has proposed 5 biotypes based on the characteristics listed in Table 3,[32] on the following page. Heddleston obtained the results tabulated below with cat, dog and human cultures in the sugars listed in Table 3.[62]

Source		Mannitol	Sorbitol	Glycerol
Human	n = 33	100%*	93.9%	78.0%
Cat	n = 22	100%	81.8%	86.4&
Dog	n = 13	30.8%	30.8%	61.4%

*Percent of strains which fermented the sugar.

He also found that humans, cat and dog cultures varied in their fermentation of dextrose, mannose, raffinose, trehalose and xylose.[62]

Heddleston and Wessman[61] did glycerol and sorbitol fermentations in a study of 30 human cultures. They found that all the cultures which were somatic types 3 and 4 fermented sorbitol and 21 out of 25 fermented glycerol. One cat and one dog bite culture (both type 3) fermented both sugars. A type 12 culture fermented glycerol but not sorbitol.[61] Smith also reported on mannitol, sorbitol and glycerol fermentation of cat and dog cultures.[129]

Antigenic Nature

Bacterial species have a variety of antigenic determinants in their cell walls and in their capsules. In many cases the chemical composition of these determinants has been analyzed. Numerous serologic and immunologic tests have been developed to demonstrate the chemical

Table 3. Proposed biotypes of P. multocida.

Biotypes	Source	Hyaluronidase	Acriflavine	Iridescence	Mannitol	Sorbitol	Glycerol	Colony
Mucoid	Poultry	+	-	+	A	V	V	Large mucoid
Hemorrhagic septicemia	Cattle & Buffalo	-	-	+	A	V	V	Medium
Porcine	Swine	-	+	+	A	V	V	Medium
Canine	Dogs	-	-	-	N	N	(N)	Small
Feline	Cats	-	-	-	(A)	(A)	(A)	Small

A - fermentation; N - no fermentation; V - variable; ( ) - most cultures.

determinants (antigens) on organisms and subsequently to identify the bacteria. In many cases, strains within a species have different antigens which can be used to identify the strains. Serologic and immunologic identification of a strain can be useful in epidemiological studies and in vaccine formulation.

Prince and Smith studied the antigenic nature of P. multocida. Antigens were released by Mickle disintegration of the cells. Eighteen antigens were identified by immunoelectrophoresis. All but the α, β and γ antigens were internal and common to all strains.[115] The α and β antigen were capsular components identical to those found by Knox and Bain.[79] The γ antigen was the strain specific somatic antigen.[115]

P. multocida has lipopolysaccharide (LPS) in its cell wall as do the enteric gram negative bacilli.[89] Several investigators have analyzed the chemical composition of LPS in P. multocida.[5,89] They used the method of Westphal et al. to extract it. Essentially this method involved heating the culture in 90% phenol at 65°C and centrifuging it. LPS was in the supernatant.[146] Bain and Knox, using Robert's Type I strain,[5] and MacLennan and Rondle[89] found that the LPS of P. multocida was chemically like that of other Enterobacteriaceae. The γ antigen of Prince and Smith was found to be LPS, also.[115]

Heddleston and Rebers demonstrated that LPS was a component of P. multocida endotoxin, using a gel diffusion precipitin test (GDPT). Free endotoxin (FET) was extracted by treating the cells with cold formalinized saline, centrifuging and purifying the FET by gel filtration. In the GDPT, LPS produced a line of partial identity with the FET. Heddleston and Rebers analyzed the composition of FET as follows:

it had a molecular weight of 7.9 x 10^6 daltons and was 25 to 27% protein, 10 to 11% carbohydrate and 12 to 18% phospholipid. The proteins were composed of 18 amino acids. The carbohydrates included glucose, galactose, heptose and hexosamine. The lipids included phosphatidylethanolamine and the fatty acids, myristic, palmitic and palmitoleic acids. FET differed from LPS in that it contained more protein and was immunogenic.[60]

Heddleston et al. found that a heat stable antigen, one not destroyed by boiling, was present in P. multocida.[58] Heddleston and Rebers showed that the heat stable antigen and the FET were identical by GDPT and slightly different by immunoelectrophoresis. The heat stable antigens can be used to identify serotypes of P. multocida.[60]

In 1928, Hoffenreich first described a capsular antigen on P. multocida. He found it to be polysaccharide in nature.[65] Since then several investigators have studied the chemical composition of the capsule. Knox and Bain[79] and Prince and Smith,[115] using Robert's Type I (Carter's type B) strain,[120] removed the capsule with saline and separated the components by isoelectric precipitation. The precipitate or the α fraction, as Prince and Smith named it, contained protein; the supernatant or β fraction contained polysaccharide. The polysaccharide contained fructose and hexosamine. Iridescent and blue variants of the same strain were studied. They produced the same kind of capsule but the blue variant had insufficient protein to keep it attached to the cell. Carter and Annau also analyzed the capsular composition of a type B strain of P. multocida. They studied mucoid, iridescent and intermediate dissociation variants of this strain and determined the

percentage of nitrogen, phosphorus and reducing substances present.[23] The capsule of the mucoid type A strain was composed of hyaluronic acid as demonstrated by its depolymerization by hyaluronidase.[31] The type D capsule was thought to be a highly acidic polyelectrolyte of N-acetylgalactosaminuronic acid: this was because the Vi antigen of Salmonella typhi produced the same reaction in acriflavine as did type D P. multocida and was composed of the uronic acid named above.[30] More recently Mukkin and Nilakantan extracted the capsule from Robert's type I strain. Using potassium thiocyanate, they identified the sugars and the amount of protein composing the capsule.[97]

Serotyping

Before the chemical nature of the antigens of P. multocida was determined, a number of serotyping systems had been developed. Rosenbusch and Merchant,[124] Little and Lyon[88] and Roberts[120] have reviewed the early studies. Among the early serotyping systems, each of which employed a different type of serologic test, were the following.

In 1929, Cornelius[37] and in 1934, Ochi[106] divided the species into 4 groups based on agglutination of the bacteria in "type specific" antisera which had been absorbed by heterologous types. In 1935, Yusef, using an acid extracted antigen, classified the species into 4 groups by a precipitin-absorption technique.[149] In 1936, Khalifa developed 2 immunotypes based on active and passive immunization. He correlated these with 3 fermentation patterns.[76] In 1943, Little and

Lyon divided the species into 3 groups, using a slide agglutination technique, the results of which correlated with those obtained by passive immunization.[88] In 1947, Roberts classified P. multocida into 4 groups on the basis of mouse protection tests. This involved injecting the antiserum subcutaneously into a mouse and 24 hours later injecting the bacteria intraperitoneally. Roberts type I has been associated with hemorrhagic septicemia and type II mainly with avian sources.[120]

Most early serotyping systems did not distinguish between capsular or somatic antigens. At first capsular typing was done because many strains were found to have common somatic antigens.[101] This was initially done by precipitin and capsular swelling tests. By these methods, Carter identified 4 types which he named A, B, C and D. Types B and D were found to be identical to Roberts' types II and IV, respectively.[24] Since encapsulated strains may dissociate into nonencapsulated variants, precipitin and capsular swelling tests are unreliable. Polysaccharide will adhere to red blood and antibodies to the polysaccharide will agglutinate the blood cells.[81] Carter applied this principle to capsular typing, using the following method. A culture, confluently growing on blood agar, was suspended in saline. This suspension was incubated at 56°C to remove the capsular material and the supernatant was incubated with human type O red blood cells. Hemagglutination in specific antiserum indicated the type of capsule present.[24] Namioka and Murata developed a slide agglutination test for capsular typing. Only cultures consisting of iridescent colonies were used. The cells were mixed on a slide with diluted "type specific" antiserum. Agglutination indicated the type capsule present.[99]

In 1960, cultures of P. multocida causing hemorrhagic septicemia but possessing a capsule different from type B were found in Central Africa.[27] Carter demonstrated by mouse protection[28] and indirect hemagglutination tests[27] that these cultures differed from type B. The new capsule was named type E. Penn and Nagy studied the somatic and capsular antigens of types B and E using immunodiffusion and immuno-electrophoresis.[112] Subsequent studies failed to confirm that type C represented a bona fide distinct capsular type. It was dropped as a capsular type. At present the Carter capsular types include types A, B, D and E.[103]

Other techniques have been developed to identify specific capsular types. Type A cultures were identified by the reduction of the large hyaluronic acid capsules in the presence of staphylococcal hyaluronidase. This was demonstrated by making parallel streaks of P. multocida cultures on blood agar and by streaking a hyaluronidase-producing Staphylococcus aureus at right angles to them. If the culture had a type A capsule, the mucoid character of the growth and thus, the colony size was diminished close to the staphylococcal streak.[31] Type D capsules were identified by the manner in which the bacteria settled in 0.1% acriflavine. Strains with a type D capsules developed a coarse (floccular) precipitate in 5 minutes which settled as a heavy precipitate in 30 minutes. Strains with other types of capsules settled more slowly and only as a fine precipitate.[30] Strains with type B and E capsules have been identified by a coagglutination test. In this test, antibodies to type B or E capsular material were bound by their F_c fragment to the A protein on S. aureus. In the presence of a culture

with a homologous capsule, the S. aureus and P. multocida coagglutinated.[118]

It became apparent that serotyping systems based solely on mouse protection and capsular typing were inadequate. Namioka and Murata,[100] and Heddleston et al.[58] have developed serotyping systems based on somatic antigens. Namioka and Murata serotyped cultures using a tube agglutination test. Cultures were treated with 1 N HCl which was thought to expose the somatic antigen. Antisera made from antigen prepared in the manner above were absorbed with heterologous strains. These were employed in a tube agglutination test. Using this method, Namioka divided P. multocida into 6 somatic types. Types 1, 5 and 6 had subtypes.[100] Later, when he combined capsular types with somatic types, the number of serotypes increased to 10 in 1962[102] and 12 in 1971.[103]

Heddleston et al. serotyped cultures by the heat stable antigen identified in the GDPT. The antigen was prepared by growing the culture on dextrose starch agar and boiling it in an 8.5% NaCl, phosphate solution. The supernatant contained the heat stable antigen. The antisera were prepared in chickens by injecting a formalinized whole cell preparation subcutaneously. The somatic type of a culture was indicated by a precipitin line between the heat stable antigen and "type specific" antiserum in the GDPT.[58] During the time since the test was developed, 16 serotypes have been found.[16,19,55,57,59]

One of the reasons for developing serotyping systems was to do seroepidemiologic surveys. Using the Heddleston system, studies of the somatic types of P. multocida cultures from animal and human sources have been done. From 1971 through 1973, Blackburn et al.

tabulated the serotypes of 762 cultures from 20 animal species collected nationwide. The predominate serotypes were types 1, 3 and 4, accounting for 53% of the cultures. In this survey, Blackburn et al. found the serotypes tabulated below among the human isolates:

Somatic type	1	3	3-4*	4	4-7*	6	12	13
Number of cultures	1	14	5	3	4	1	1	1

*Culture reacted with 2 antisera.

In the same survey, isolates from cats included one each of types 3, 4 and 5.[16] Heddleston and Wessman serotyped 30 human cultures, collected primarily in Iowa. They obtained the results tabulated below from the survey.[61]

Somatic types	1	3	3-4*	4	6	12	13	Untypable
Number of cultures	1	17	1	7	1	1	1	1

*Culture reacted equally with 2 antisera.

Cross reactions can occur between different serotypes and immunotypes. This can be demonstrated in a number of ways. Antiserum to one strain may protect against the challenge of another strain in the mouse protection test.[115] In a gel diffusion test, whole cell extracts may react with more than one "type specific" antiserum.[16] Some of the pairs of antisera with which Blackburn et al.[16] and Heddleston et al.[56,61] found one strain to have reacted are the following: types 1&3, 2&5, 2&12, 3&4, 3&11, 3&12, 3, 4&12, 4&5, 4&7, 4&12, 7&10, 7&11 and 7&12.[100] In addition, absorption of an antiserum by a heterologous strain may remove cross reacting antibodies.

Seroepidemiologic studies have been carried out to determine the capsular types of animal and human isolates. Strains with type A capsules were widely distributed. The strains causing fowl cholera were generally type A. Strains with type B and E capsules were associated with hemorrhagic septicemia in cattle and water buffaloes.[24] Type D strains occurred sporadically but had a wide host range, being particularly common in swine.[103] Cat and dog strains often had no capsule, producing blue colonies which tended to autoagglutinate.[32] Carter and Bain reviewed studies which documented the capsular type of human cultures. Of 47 cultures, there were 20 type A's, 11 type D's and 16 nontypable. In another review,[25] Carter documented 3 type A's, 4 type D's and 3 that were untypable. Two of the type A's were from cat bites or scratches and 3 of the type D's were from respiratory infections.[26]

Namioka and Murata, using Carter's capsular types and his own somatic types, identified each strain as a serotype. They were designated with the number of the somatic type followed by a colon and then the capsular type, e.g., 1:A, 6:B, etc.[100] Certain serotypes were the primary cause of disease in specific animals. Some examples were 5:A and 8:A caused fowl cholera, 6:B and 6:E caused hemorrhagic septicemia in cattle and 1:A and 2:D caused pneumonia in swine.[103] In an extensive study of 156 cultures from many sources, 2 human cultures were isolated. A 5:- and an 8:- were isolated from wounds. Also, a 3:D and 8:- were isolated from cats with pneumonia.[102]

Brogden did a comparative study of 5 serotyping systems to determine the correspondence of the types among the systems. The systems

were those of Roberts, Little and Lyon, Carter, Namioka and Heddleston. One example of correlation between serotypes is that Namioka's 5:A and 8:A are equivalent to Heddleston's types 1 and 3 respectively.[20]

Ecology

P. multocida is frequently a commensal which may cause disease under certain circumstances. It may be a part of the normal flora of the upper respiratory and digestive tracts of many animals. This was first discovered in 1895 when it was isolated from the mucous membranes of healthy animals.[94] P. multocida has been recovered from apparently normal animals in the following percentages: 5 to 9% of piglets from their nasal secretions,[54] 26% of pigs from their lungs, 51% of pigs from their mouths,[128] 6 to 8% of cattle,[29] 3 to 5% of buffalo,[128] 40% of sheep,[29] 10% of dogs from their nostrils, 54% of dogs from their tonsils, 90% of cats and 14 to 17% of rats.[128] It has also been isolated from a wide variety of wild animals and birds.[29,62]

Animal Diseases

As its name suggests, P. multocida causes disease in many animal species.[33] Some strains are more virulent than others. Virulence can, however, be increased by passage of the organism in such animals as cattle, rabbits, mice and chicken embryos. The disease process can take acute, subacute and chronic forms. The acute form is characterized particularly by vascular congestion and serous or subserous hemorrhages. Hemorrhagic lesions also characterize the subacute form. In the

chronic form, areas of necrosis and abscess formation may be present in various tissues and organs accompanied usually by a progressive loss of condition. P. multocida can be either a primary or secondary cause of disease.[33]

Pasteurellosis varies somewhat in different animal species. Chickens and turkeys have a primary pasteurellosis called fowl cholera. It is seen in all 3 forms although the chronic form is the most common. A secondary infection may occur involving air sacs and sinuses. Other birds such as domestic, wild and eider ducks,[123] geese and pheasants[71] may also contract fowl cholera.

P. multocida causes hemorrhagic septicemia in cattle and water buffaloes. This is an acute disease of great economic importance in Africa, the Near and Middle East and South East Asia. It does not occur in North and South America except occasionally in American bison.[33]

Cattle and sheep are subject to a pneumonic pasteurellosis which is a worldwide disease of economic importance. It is a bronchopneumonia usually referred to as shipping fever. Pneumonic pasteurellosis is mainly precipitated by stresses incidental to shipping and crowding and caused by secondary invasion of P. multocida or P. hemolytica.[33]

Other domestic animals contract primary pasteurellosis but more commonly they acquire the secondary form of the disease. Among these animals are swine and to a lesser extent horses, cats, dogs and many wild animal species. Pasteurellosis in swine is called enzootic pneumonia and the primary cause is a mycoplasma. Cats and dogs occasionally get secondary infections due, no doubt, to the high carrier rate of P. multocida in these animals. P. multocida is one of the

causes of snuffles in rabbits which is primarily a disease of the upper and lower respiratory tract. Suppurative metastases are frequent and fatal septicemias are not uncommon.[33]

P. multocida has been associated with many infections other than those involving the respiratory system. These infections include such diverse conditions as abscesses, mastitis, abortion, encephalitis,[29] conjunctivitis[121] and animal bite infections. P. multocida has also been associated with disease in many wild animal species.

As a result of the extent and importance of disease caused by P. multocida a great deal of research has been done over the years on the development of vaccines for their prevention. In fact, the first vaccine tested by Louis Pasteur was a live vaccine for the prevention of fowl cholera.

Human Diseases

Although P. multocida is a commensal of numerous animal species, it is thought to rarely be a part of the normal flora of humans. In a study of 71 veterinary students, Smith found two who carried P. multocida in their throats.[130] Humans can, however, become infected with P. multocida when they come in contact with infected or carrier animals. Infections take 2 principal forms. These are skin or wound infections, and internal infections which involve various organs of the body, most commonly the lungs. Numerous papers have been written documenting human infections. These papers include information on underlying physiological conditions, clinical symptoms, treatment, sequelae, association

with animals and occasionally references to biotypes or serotypes. The first documented case of human infection with P. multocida was described by Brugnatelli in 1913.[21] It was a case of puerperal sepsis in a farmer's wife. As more people learned how to identify P. multocida, more cases were recognized.

Skin Infections

The first type of infection is a skin lesion resulting from trauma. Animal bites and scratches are the most common cause, cats and dogs being the primary animals. Kapel and Holm in 1920 were the first to document a case of P. multocida infection following a dog bite.[74] In 1937, Rimpau described 3 cases of wound abscesses resulting from cat bites.[119] He collaborated with Schenk who did the biochemical studies and also isolated P. multocida from cats.[125] Many articles have been written describing cases of cat and dog bites infected with P. multocida.[39,66,67,72,93,139]

The following is the typical clinical picture seen in a bite infected with P. multocida. Symptoms of pain,[75,139] redness[74] and edema develop[1,46,75] rapidly, sometimes within 24 hours. A gray serosanguineous[47] or yellow-green purulent discharge is produced[1,46,72] and the area becomes abscessed and necrotic.[139] The infection can also take the form of a cellulitis and spread to regional lymph nodes.[1,17,139] (The abscessed area is slow to granulate and heal.[1,17,42,139]) Jones and Smull reported a case of thrombophlebitis near the site of a cat bite and subsequent pulmonary embolism.[72]

Treatment of these wounds requires intensive antibiotic therapy, in higher doses than usual,[42] immobilization, hot pads and for the more

severe cases, debridement and drainage. Although it is gram negative, penicillin is the drug of choice for the treatment of P. multocida infections.[72] In many of the case studies, the antibiotic sensitivity pattern of the strain was given. Recently, Stevens et al. have determined the minimum inhibitory concentrations (MIC) of a number of drugs for P. multocida. Penicillin, ampicillin, carbenicillin, cephalothin, tetracycline and chloramphenicol had the lowest MIC's.[135] The wounds heal slowly and may leave nerve, tendon or muscle damage.[45,75] Those with this skin infection may experience systemic symptoms such as fever and malaise.[140]

Wound infections with P. multocida are usually the result of cat or dog bites or cat scratches although bites from other animals such as a lion, panther, rabbit,[63] oppossum and rat have been implicated.[68] Wounds infected in other unique ways have been documented. These have included hand ulcers from milking cows with ulcerated udders,[93] cuts contaminated while dressing a turkey,[69] facial injuries from the kick of a horse,[104] cuts from infected trapping lines,[67] injuries received while falling off a bicycle[93] and a forearm wound punctured with a rake.[69]

In 2 studies, all of the animal bite cases over a period of time were used to calculate various statistical parameters. Hubbert and Rosen documented 180 cases during 1965 to 1968, from all parts of the United States. Statistics on age, sex and type of animal were presented. The region of the body where the bites occurred was also given. The upper extremities predominated. The animal bites occurred throughout the year with the largest number occurring from July through

September.[68] Francis et al. presented data on 48 animal bites which occurred in Portland, Oregon. They presented the same kind of data as the first study. In addition, they listed the type of symptoms which occurred in wound infections and the number of cases in which they occurred. They also indicated the distribution of cat and dog bites. Cat bites comprised 76% of the bite infections and dogs, 24%.[47]

While most studies indicated the general characteristics of P. multocida, only a few described the unique characteristics of cat and dog strains. Smith found that P. multocida strains from cat and dog bites were non-encapsulated, had small non-mucoid, non-iridescent colonies, were acid agglutinable and were moderately pathogenic for mice. Cat strains fermented mannitol and sorbitol while dog strains were variable.[130] These facts correlated with Smith's[129] and Carter's[132] findings on cultures isolated from cats and dogs.

Internal Infections

The internal infections are secondary to a primary wound or respiratory infection usually presumed to spread hematogenously from the primary to the secondary site or sites. Exceptions are eye infections which are usually the result of direct trauma to the eye. Internal infections include such diverse infections as pneumonia and sinusitis, osteomyelitis and arthritis, otitis media, meningitis and brain abscess, peritonitis, vaginitis and cystitis, ophthalmitis and septicemia.

The most common site of P. multocida infection, besides the skin at the site of trauma, is the respiratory tract. In 1919, Debré reported the first case of a respiratory infection from which P. multocida was isolated. It was found in the pharynx and empyema fluid.[40] Other early cases included the following. In 1938, Mulder isolated P. multocida from the sputum of a 15 year old with bronchitis[98]; in 1946, Ory et al. from empyema fluid[108]; in 1952, Bezjak and Mimica from the maxillary sinus[14]; in 1969, Holloway et al. from 2 cases of bronchiectasis,[66] and in 1964, Maneche from a fatal case of pulmonary abscess and also from a lung mass in a patient with emphysema.[90] Henderson has reviewed other early cases.[63] Since that time many reports of respiratory infections have appeared.[4,39,49,52,64,66,90,92,96,122,127]

In most cases of respiratory infections caused by P. multocida there was some underlying respiratory condition which predisposed to infection. Out of 80 respiratory isolates, Hubbert and Rosen related 75 to underlying respiratory conditions.[69] Among the common clinical diagnoses were bronchiectasis, bronchogenic carcinoma, chronic bronchitis, emphysema, obstructive lung disease, pneumonia, tuberculosis, tonsillitis and sinusitis.[69,107] P. multocida infection has also been associated with two cases of underlying congestive heart failure[90,122] and one case of underlying congenital respiratory and cardiac abnormalities.[52]

P. multocida can infect any area of the respiratory tract, producing sinusitis, bronchitis, pneumonia or even progress to empysema. In these cases, it can be isolated from sinus or bronchial washings, sputa, transtracheal aspirates in empyema and from fluids obtained by

thoracentesis. The following clinical symptoms may be seen in respiratory infections. The patient's sputum may be yellow-green.[35] He may have shortness of breath,[92,127] diminished breath sounds,[39,127] dullness to percussion in his chest[52,92] and bronchial obstruction.[92] Minute calcifications may be seen on chest x-rays.[51] In more severe cases, pleural effusion may develop.[52,127] Nasal infections may take the form of chronic sinusitis with a yellow green discharge.[7]

Several surveys of infections caused by P. multocida provide information on the relative number of respiratory infections, the specific underlying conditions involved and treatment. Holloway et al. found 5 respiratory and 16 wound infections from 1952 to 1959.[66] In a survey of 550 random specimens, Brodie and Henderson isolated P. multocida from 2.[19] Miller identified 20 respiratory out of 49 total infections in New York during 1956 to 1964.[93] Jones and Smull reported 5 cases of pneumonia and 2 of sinusitis during 1961 to 1971.[72] From 1965 to 1968, Hubbert and Rosen tabulated 80 respiratory cases out of 136 which were not associated with animal bites.[69]

Many people with respiratory infections caused by P. multocida had prior association with animals. Hubbert and Rosen found that 15 patients had pets and 15 more had contact with farm animals.[69] Goldenberg et al.,[52] Bartley and Hunter[7] and Bezjak and Mimica[15] associated the patients in their cases with farm animals. More specifically, Morris et al. isolated P. multocida from the sputum of an animal inspector with bronchiectasis[96]; Jones and Smull, from the sputum and bronchial washings of a butcher with pneumonia and bronchogenic carcinoma[72] and Hubbert and Rosen, from 3 people who worked in

slaughter houses. The latter study also included 2 respiratory isolates from patients associated with wild animals, one of whom was a fox farmer.[69]

In the above respiratory cases, no reference was made to serotyping the strains isolated but the following biochemical and morphological observations were made. In 2 cases the colonies were described as large, mucoid and viscous, indicating that the strains were probably encapsulated. In 2 cases, biochemical reactions that might define a biotype were given.[36,96] In the case of Bezjak and Mimica, the strain did not ferment sorbitol or glycerol[15] and in the case presented by Brodie and Henderson, the strain fermented mannitol and sorbitol but not glycerol.[19] Smith did a study of cultures isolated from humans, 6 of which were isolated from internal infections and 2 from the throats of healthy individuals. Two resembled feline strains, 3 resembled bovine strains and 2 resembled porcine strains. They were all alike in the sugar fermentations performed. All but those resembling cats were encapsulated.[130]

Osteomyelitis is usually caused by an animal bite penetrating the periosteum or all the way to the bone proper. An abscess may develop which is slow to heal.[136] Several cases of this type have been documented in the literature.[63,66,69,136,137] In one such case, a diabetic had a stasis ulcer infected with P. multocida which developed into osteomyelitis. Septic arthritis usually develops in people with underlying joint disease. Rheumatoid arthritis or degenerative joint disease which has been repaired with a prosthetic device are most common.[113] Since patients with arthritis are often treated

with steroids, they are more susceptible to bite infections spreading to and settling in damaged joints.[91,137,145] Clinically the patient has a painful, inflamed joint[8] from which purulent fluid can be aspirated.[113] The lymph nodes may be swollen and the patient's temperature and white blood count elevated.[3] Bone infections are treated by debriding the surface of the wound,[113] aspirating purulent fluids,[3,91] treating systemically with antibiotics,[3,134] irrigating with acetic acid[3] and in severe cases, bone excision.[132] There are several cases in the literature of joints infected with P. multocida.[83,114,134,145] Spagnuolo documented 10 cases of septic arthritis, 5 of which had rheumatoid arthritis as an underlying condition. Four of the patients with rheumatoid arthritis had had steroids. In one of these cases the patient had degenerative joint disease and in 3, prosthetic devices.[134] Arvan and Victor also documented a case of a knee replacement infected with P. multocida.[3] Most of these cases were associated with animals especially cat bites.[3,10,66,72,91,134,136,145] Spagnuolo found 7 out of 10 cases associated with animals.[134] Jones and Smull documented one case of a dairy farmer with a septic shoulder joint.[72]

Meningitis and brain abscesses caused by P. multocida can result from a skull fracture, brain surgery, an animal bite to the head or otitis media. Controni and Jones reviewed the literature up to 1964, documenting 14 cases of meningitis worldwide. Five were the result of a skull fracture, 2 were subsequent to brain surgery and 3 were associated with animals.[36] Since that time other cases have appeared in the literature.[15,78,142,148] There were 4 cases of meningitis in young children, all of whom had contact with family dogs.[15,78,142,148]

There were 2 other cases of meningitis following trauma to the head.[69,84] In more severe cases brain abscesses have developed.[78,83,86,93] Cases of otitis media have been reported. These are included with the meningitis and brain abscess cases because the infection can spread contiguously from the inner ear into the meninges. Clinically, meningitis caused by P. multocida resembles other septic meningitis in which the spinal fluid has increased white blood cell counts and protein concentration and decreased glucose concentration.[69,83,93] In many of the above cases the patients have been associated with animals such as a pet hen,[36] cats,[86] dogs,[15,78,148] and infected rabbit muscle, used as a hemostat during a craniotomy.[63]

The second most common internal infection involves the gastrointestinal tract. In 1917, Van Boer documented the first case of enteritis. The man in this case worked where there were chickens with fowl cholera. Gastrointestinal infections can take several forms.[144] One common type is appendicitis[69,72] of which Henderson documented several early cases.[63] Other types were infected abdominal surgical sites,[69] peritonitis[51,69,70,93] and gastroenteritis.[63,69,93,144] As with other internal P. multocida infections, people with underlying physiological conditions are predisposed to infection. Alcoholism, gallbladder surgery,[51] cirrhosis or chronic liver disease predispose to peritonitis.[51,70] In some of these cases, association with cats or dogs has been suggested.[51,70] Hubbert and Rosen found 13 out of 24 abdominal cases associated with pets or farm animals.[69] In one case, of Gèrding et al., the patient was a butcher.[51]

Occasionally urinary or vaginal infections have occurred. Urinary infections are usually secondary to a urinary tract abnormality such as dysuria, chronic cystitis,[6,69] obstructive uropathy,[6,80] carcinoma[54,80] or neurogenic bladder.[80] The non-urinary conditions, diabetes and rheumatic heart disease, have been identified as predisposing factors in urinary tract infections.[80] P. multocida with a type A capsule was isolated from a urinary tract infection of a patient with metastatic cervical carcinoma.[41]

There have been a few reports of eye infections. Levy-Bruhl documented several early cases.[85] More recently, Hubbert and Rosen found 4 cases of conjunctivitis; in one the infection had spread from a sinus. In 3 of these cases animal contact was noted; one patient had worked in a meat rendering plant.[69] There have also been 2 cases of corneal ulcers following cat scratches to the eye. The clinical signs in these 2 cases were pain, edema and decreased vision with eventual scarring.[50,117]

Septicemia may be a sequela to a localized P. multocida infection. It has most commonly occurred along with peritonitis subsequent to cirrhosis or alcoholic jaundice.[9,70,105,111,147] Septicemia has also accompanied endocarditis associated with rheumatic heart disease,[2,82] meningitis,[15,142,148] septic arthritis[113] pneumonia[72] and wound debridement and drainage.[47] Most of these patients had associated with dogs[15,70,111,142,148] or cats.[9,105,113] The capsular types of 2 of the organisms isolated from septicemias were identified. A type A capsule was found on the P. multocida culture from a 65 year old with Laennec's cirrhosis.[111] A P. multocida-like organism with a type E capsule was

isolated from a 10 year old East African with subacute bacterial endocarditis and rheumatic heart disease. There was some question about the identification of this culture. This was because it grew on MacConkey agar, was indole negative and the capsule was determined by slide agglutination.[82]

This literature review has attempted to give some background for the subsequent study. It has described some of the taxonomic, biochemical and morphologic properties of P. multocida. It has briefly explained part of the chemical nature of the organism and some of the capsular and somatic typing systems used to identify different strains of P. multocida. It has also shown that P. multocida is a commensal in many animal species but also causes disease in man and animals. Case studies of human infections of different parts of the body were documented.

MATERIALS AND METHODS

Cultures. These included 51 isolates from human infections, 10 from cats and 5 from dogs. The isolates were identified as P. multocida if they met the following criteria: gram negative coccobacillus; A/A on TSI; no growth on MacConkey agar; nonhemolytic; catalase, oxidase and indole positive; reduction of NO_3 to NO_2; fermentation of glucose and sucrose but not lactose or maltose; non-motile and urease negative. The cultures came from the donors listed in Table 4, on the following page. Information on the anatomical site of infection or lesion and animal source was recorded.

Antisera. Heddleston's 16 serotypes of P. multocida were used to prepare antisera.[16] Confluent 18 h cultures on dextrose starch agar (Difco) were washed off with 0.3% formalinized saline and diluted to a MacFarland #10. Suspensions were incubated at 37°C overnight and checked for sterility in brain heart infusion semi solid. Equal volumes of this suspension and Freund's incomplete adjuvant were thoroughly emulsified to prepare the vaccine. The antisera were produced in chickens after a single inoculation of 1 ml of the vaccine subcutaneously in the neck. After 3 weeks blood was collected by cardiac puncture and the serum recovered.[58]

Antigen. Confluent 18 h cultures on dextrose starch agar with 0.3% yeast extract were suspended in 1 ml of a solution containing 8.5%

Table 4. Cultures of P. multocida, listed by source.

Number of Cultures	Host	
20	Human	Michigan Department of Public Health Lansing, Michigan
10	Human	Edward W. Sparrow Hospital Lansing, Michigan
5	Human	Center for Disease Control Atlanta, Georgia
6	Human	Children's Hospital Buffalo, New York
2	Human	Grant Hospital Columbus, Ohio
2	Human	Ingham Medical Center Lansing, Michigan
3	Human	Veterinary Research Institute Ipoh Perah, Malayasia
2	Human	Children's Hospital Detroit, Michigan
1 2	Human Cat	Ottawa General Hospital Ottawa, Ontario, Canada
8 5	Cat Dog	Veterinary Clinic Michigan State University East Lansing, Michigan

NaCl, 0.02 M phosphate (0.414 g $NaH_2PO_4 \cdot H_2O$ and 4.79 g $Na_2HPO_4 \cdot 7H_2O$) and 0.3% formalin. The suspension was vortexed, boiled for 1 h and centrifuged at high speed for 15 min. The supernatant contained the heat stable antigen.[58]

Gel diffusion precipitin test (GDPT). The agar was composed of 0.9% Special Agar-Noble (Difco), 8.5% NaCl and 0.01% merthiolate. Five ml of agar was placed in a tightly covered petri plate, 5 cm in diameter. Wells, 4 mm in diameter, were cut equidistant around a central well at a distance of 6 mm from center to center. Antigen was put in the central well and antisera in the surrounding wells. The plates were incubated at 37°C for 1 or 2 days. Lines of precipitation were observed by placing the plates over a light source and using a magnifying glass. Location, intensity and breadth were noted. If the antigen reacted with several antisera, the test was repeated with all the reacting sera.[58]

Serotyping. The somatic type was defined by the type of antiserum with which the strain reacted most intensely. The type of the next strongest reacting antiserum was placed in parenthesis to indicate a "secondary" cross-reacting type.

Capsular typing. Type A cultures were identified by the hyaluronidase test. Blood agar, freshly prepared or moistened with brain heart infusion broth, was inoculated with parallel streaks of P. multocida cultures. A hyaluronidase-producing Staphylococcus aureus was streaked at right angles to the P. multocida streaks. Colonies of cultures with type A hyaluronic acid capsules were reduced in size in

the vicinity of the S. aureus streak.[31]

Cultures with type D capsules were identified by the acriflavine test. Overnight BHI cultures were centrifuged and resuspended in 1 ml of BHI. One-half ml of the suspension was mixed with 0.5 ml of 0.1% acriflavine solution. Type D cultures produced a coarse flocculation in 5 minutes.[30] Known type A and D cultures were also included as controls.

Cultures found to lack capsular substance by the above methods were passed in mice to induce capsule formation as follows: a Swiss albino mouse was inoculated intraperitoneally with 0.3 ml of an 18 h broth culture. After the mouse expired, the heart blood was cultured.

Colony variants. Isolated colonies on blood agar were observed for size (small, medium or large) and consistency (dry, pasty, creamy, smooth or mucoid). Isolated colonies on dextrose starch agar were observed by obliquely transmitted light for iridescence. The colonies were described as blue, iridescent or intermediate.

Fermentation studies. Each culture was inoculated into the 1% phenol red sugars, mannitol, sorbitol and glycerol. A yellow color was interpreted as fermentation of the sugar; no color change as no fermentation.

RESULTS

The results of the examination of the 66 cultures are listed in Table 5. The human cultures are grouped by the specimen from which they were isolated. Cat and dog cultures are grouped together at the end of the table. The data given includes animal association, the somatic and capsular type, fermentation reactions in mannitol, sorbitol and glycerol and colonial variant, including size, consistency and optical property.

The number of cultures of each "primary" serotype are listed in Table 6. The primary serotype was the type of the antiserum with which the culture reacted most strongly. Among the human cultures, types 1 and 3 predominated. There were 5 type 4 cultures and a few each of types 5, 8, 11 and 12. Among the cat cultures, type 3 predominated with types 1, and also 12 represented. The dog cultures included types 1, 3 and 8. Human cultures had the same somatic types as the animal cultures with 2 exceptions. Types 4 and 11 were only found among human cultures.

The cultures are listed by source and serotype in Table 7. In the lower part of the table the sources are classified as either wound or internal infections. There were 22 cultures from wounds and 26 from internal infections. Cultures were from different internal sites with 14 from the respiratory tract and 5 from blood. A number of the same serotypes were isolated from both wounds and internal infections

Table 5. Results of the examination of human, cat and dog cultures of P. multocida.

Source	Animal Association	Serotype	Fermentations			Colony variant, Size, Consistency, Optical variant
			Mannitol	Sorbitol	Glycerol	
Human Cultures						
Wound	Dog	-:1	N	N	N	Small, Pasty, Blue
Bite	Dog	-:1	N	N	N	Small, Pasty, Blue
Bite	Dog	-:1(5)	A	A	N	Medium, Pasty, Blue
Bite	Dog	-:1(7)	N	N	N	Small, Dry, Blue
Bite	Dog	-:3	A	A	A	Medium, Creamy, Blue
Bite	Dog	-:4(3)	A	A	A	Small, Creamy, Intermediate
Bite	Dog	-:11	N	N	N	Small, Pasty, Blue
Bite	Cat	-:1	N	N	N	Small, Pasty, Blue
Bite	Cat	-:1	A	N	N	Small, Pasty, Blue
Bite	Cat	-:1(7)	A	A	A	Medium, Creamy, Blue
Bite	Cat	-:3	A	A	A	Medium, Smooth, Blue & Intermediate
Bite	Cat	-:3(4)	A	A	A	Large, Creamy, Blue & Intermediate
Bite	Cat	-:8(5)	A	A	A	Medium, Creamy, Blue
Bite	Cat	D:8(13)	A	N	N	Medium, Creamy, Blue
Bite	Leopard	-:3(1)	A	A	A	Medium, Pasty, Blue
Bite	Unknown	-:1	A	A	A	Medium, Creamy, Blue
Abscess	Unknown	-:3	A	A	A	Medium, Creamy, Intermediate
Bite	Unknown	-:3(1)	A	A	A	Small, Creamy, Blue

continued

Table 5--continued

Source	Animal Association	Serotype	Fermentations			Colony variant, Size, Consistency, Optical variant
			Mannitol	Sorbitol	Glycerol	
Human Cultures--con'd.						
Bite	Unknown	-:3(1)	A	A	N	Medium, Creamy, Blue
Wound	Unknown	A:3(4)	A	A	A	Large, Mucoid, Intermediate
Bite	Unknown	-:5(1)	A	A	A	Small, Creamy, Blue
Sputum	Unknown	-:1	A	A	A	Small, Pasty, Blue
Sputum	Unknown	-:1	A	N	N	Medium, Creamy, Blue
Sputum	Unknown	A:1(5)	A	A	A	Large, Mucoid, Iridescent
Sputum	Unknown	-:3	A	A	A	Small, Creamy, Blue
Sputum	Unknown	A:3	A	A	N	Large, Mucoid, Iridescent
Sputum	Cat	D:3(1)	A	A	A	Large, Mucoid, Intermediate
Sputum	Unknown	A:3(4)	A	A	N	Large, Mucoid, Iridescent
Sputum	Unknown	-:4(7)	A	A	A	Small, Creamy, Blue
Sputum	Unknown	A:4(12)	A	A	A	Large, Mucoid Iridescent
Bronchial washing	Unknown	-:1	A	A	A	Medium, Smooth, Blue
Bronchial washing	Unknown	-:3	A	A	N	Small, Creamy, Blue
Naso-trach	Unknown	-:3(4)	A	A	N	Medium, Smooth, Blue
Pleural fluid	Unknown	-:1	N	N	N	Small, Creamy, Blue

continued

Table 5--continued

Source	Animal Association	Serotype	Fermentations			Colony variant, Size, Consistency, Optical variant
			Mannitol	Sorbitol	Glycerol	
Human Cultures--con'd.						
Throat	Unknown	-:5	A	A	N	Medium, Smooth, Blue
Spinal fluid & Blood	Dog	-:3	A	A	A	Medium, Creamy, Intermediate
Spinal fluid	Unknown	-:5	A	A	A	Medium, Creamy, Intermediate
Blood	Unknown	A:1(4)	A	A	N	Large, Creamy, Iridescent
Blood	Unknown	A:3	A	A	N	Large, Mucoid, Iridescent
Blood	Unknown	A:3(1)	A	A	A	Large, Mucoid, Iridescent
Blood	Unknown	A:4(3)	A	A	A	Large, Mucoid, Intermediate
Joint	Unknown	-:1(4)	A	A	A	Small, Pasty, Blue
Pelvic abscess	Unknown	A:3(4)	A	A	A	Large, Mucoid, Intermediate
Liver abscess	Unknown	A:3(4)	A	A	N	Large, Mucoid, Intermediate
Postoperative wound	Unknown	A:12(4,3)	A	A	A	Large, Mucoid, Iridescent
Eye	Unknown	A:4(3)	A	A	N	Medium, Creamy, Iridescent
Ear	Unknown	A:1	A	N	A	Large, Mucoid, Intermediate
Human	Unknown	-:3(1)	A	A	A	Medium, Creamy, Blue

continued

Table 5--continued

Source	Animal Association	Serotype	Fermentations			Colony variant, Size, Consistency, Optical variant
			Mannitol	Sorbitol	Glycerol	
Human Cultures--con'd.						
Human	Unknown	-:1(4)	A	A	A	Medium, Creamy, Blue
Human	Unknown	A:3(14)	A	A	A	Medium, Mucoid, Iridescent
Human	Unknown	A:12(1,4)	A	A	N	Large, Mucoid, Intermediate
Cat Cultures						
Skin abscess	Cat	-:1	A	A	A	Medium, Creamy, Blue
Unknown	Cat	-:1(5)	A	A	N	Small, Pasty, Blue & Intermediate
Buccal	Cat	-:1(5)	A	A	N	Small, Pasty, Blue
Nasopharyngeal	Cat	-:3	A	A	A	Medium, Pasty, Blue
Unknown	Cat	-:3	A	A	A	Medium, Pasty, Blue
Unknown	Cat	-:3	A	N	A	Small, Dry, Blue
Unknown	Cat	D:3	A	A	A	Medium, Creamy, Intermediate
Abscess	Cat	-:3(1)	A	N	A	Medium, Creamy, Blue
Unknown	Cat	D:3(1)	A	A	A	Medium, Creamy, Blue

continued

Table 5--continued

Source	Animal Association	Serotype	Fermentations			Colony variant, Size, Consistency, Optical variant
			Mannitol	Sorbitol	Glycerol	
Dog Cultures						
Unknown	Dog	-:1	N	N	N	Medium, Pasty, Blue
Lung	Dog	-:1	N	N	N	Medium, Pasty, Blue
Lung	Dog	-:3(1)	A	A	A	Medium, Pasty, Blue
Nasal fluid	Dog	-:3(1)	A	A	A	Small, Creamy, Blue
Unknown	Dog	-:8	N	N	N	Small, Dry, Blue

Note: A - fermentation; N - no fermentation.

Table 6. Number of primary serotypes of *P. multocida* by animal origin.

Humans		Cats		Dogs		
Type	Number	Type	Number	Type	Number	Total
1	17	1	3	1	2	22
3	21	3	6	3	2	29
4	5					5
5	3					3
8	2			8	1	3
11	1					1
12	2	12	1			3
Total	51		10		5	66

Table 7. Distribution of serotypes of Pasteurella multocida by source.

Source	Somatic Type																		Total
	1	1(4)	1(5)	1(7)	3	3(1)	3(1,4)	3(4)	4(3)	4(7)	4(12)	5	5(1)	8(5)	8(13)	11	12(1,4)	12(4,3)	
Dog bite	2		1	1	1				1							1			7
Cat bite	2			1	1			1						1	1				7
Wound	1				1	3		1					1					1	8
Respiratory	4		1		3	1		2		1	1	1							14
Eye									1										1
Ear	1																		1
Joint		1																	1
Pelvic abscess								1											1
Liver abscess								1											1
Spinal fluid					1							1							2
Blood		1			2	1			1										5
Unknown		1				1	1										1		4
Total	10	3	2	2	9	6	1	6	3	1	1	2	1	1	1	1	1	1	52
	1	1(4)	1(5)	1(7)	3	3(1)	3(1,4)	3(4)	4(3)	4(7)	4(12)	5	5(1)	8(5)	8(13)	11	12(1,4)	12(4,3)	Total
Wounds	5		1	2	3	3		2	1				1	1	1	1			21
Internal infections	5	2	1		6	2		4	2	1	1	2						1	27
Unknown		1				1	1										1		4
Total	10	3	2	2	9	6	1	6	3	1	1	2	1	1	1	1	1	1	52

with the following exceptions. Serotypes isolated only from internal infections were 1(4), 4(7), 4(12) and 12(4,3). Those only isolated from wounds were 1(7), 5(1), 8(5), 8(13), and 11.

The somatic types with which capsule types were associated are indicated in Table 8. Some cultures with primary somatic types 1, 3, 4 and 12 had type A capsules. The 2 cultures with type D capsules were somatic types 3(1) and 8(5). Most types 5, 8 and 11 did not have capsules.

The sources from which encapsulated cultures were isolated and the number of cultures isolated are given in Table 9. Of the 21 cultures from wound infections, one had a type A capsule as did 15 of the 27 cultures from internal infections. Within the group of cultures from internal infections, 4 of the 14 respiratory isolates, 4 of the 5 blood isolates and all of the remaining cultures from internal organs were type A. None of the cultures from cats or dogs had type A capsules. Two human and 2 cat cultures had type D capsules. One human culture was from a wound and one was from sputum. The cats from which type D cultures were isolated were owned by the patient from whose sputum the type D culture was isolated.

In Table 10, the cultures are grouped by somatic type with human and animal cultures together. Those that were type 1 came from wound and internal infections, were associated with both cat and dog sources, had different biochemical patterns on the 3 sugars and included encapsulated and nonencapsulated strains. A type 1A culture was from the ear of a Vietnamese and a -:1(4) culture was from Malaysia. A type 1 culture was isolated from one cat, and also from 2 dogs.

Table 8. Distribution of capsular types by somatic type.

	Somatic type										
	1	1(4)	1(5)	1(7)	3	3(1)	3(1,4)	3(4)	4(3)	4(7)	4(12)
Type A	1	1	1		2	1	1	4	2		1
Type D					1	2					

	Somatic type (cont'd)							
	5	5(1)	8(5)	8(13)	11	12(1,4)	12(4,3)	Total
Type A						1	1	16
Type D			1					4

Table 9. Distribution of capsular types by sources of specimen.

Source (number)	Type A	Type D
Wounds (21)	1	1
Respiratory (14)	4	1
Blood (5)	4	
Pelvic abscess (1)	1	
Liver abscess (1)	1	
Postoperative wound (1)	1	
Eye (1)	1	
Ear (1)	1	
Unknown (4)	2	
Cat (10)	0	2
Dog (5)	0	0

Table 10. Cultures of P. multocida grouped by serotype.

Source	Animal	Serotype	Fermentations		
			Mannitol	Sorbitol	Glycerol
Bite	Dog	-:1	N	N	N
Bite	Dog	-:1	N	N	N
Bite	Cat	-:1	N	N	N
Bite	Cat	-:1	A	N	N
Pleural fluid	Unknown	-:1	N	N	N
Sputum	Unknown	-:1	A	N	N
Sputum	Unknown	-:1	A	A	A
Bronchial washing	Unknown	-:1	A	A	A
Bite	Unknown	-:1	A	A	A
Ear	Unknown	A:1	A	N	A
Joint	Unknown	-:1(4)	A	A	A
Human	Unknown	-:1(4)	A	A	A
Blood	Unknown	A:1(4)	A	A	N
Bite	Dog	-:1(5)	A	A	N
Sputum	Unknown	A:1(5)	A	A	A
Bite	Dog	-:1(7)	N	N	N
Bite	Cat	-:1(7)	A	A	A
Cat	Cat	-:1	A	A	A
Cat	Cat	-:1(5)	A	A	N
Cat	Cat	-:1(5)	A	A	N
Dog	Dog	-:1	N	N	N
Dog	Dog	-:1	N	N	N
Bite	Dog	-:3	A	A	A
Bite	Cat	-:3	A	A	A
Abscess	Unknown	-:3	A	A	A
Sputum	Unknown	-:3	A	A	A
Spinal fluid and blood	Dog	-:3	A	A	A
Bronchial washing	Unknown	-:3	A	A	N
Sputum	Unknown	A:3	A	A	N
Blood	Unknown	A:3	A	A	N
Wound	Unknown	-:3(1)	A	A	A
Bite	Unknown	-:3(1)	A	A	A
Human	Unknown	-:3(1)	A	A	A
Bite	Unknown	-:3(1)	A	A	N
Blood	Unknown	A:3(1)	A	A	A
Sputum	Cat	D:3(1)	A	A	A
Human	Unknown	A:3(1,4)	A	A	A

continued

Table 10--continued

Source	Animal	Serotype	Fermentations		
			Mannitol	Sorbitol	Glycerol
Bite	Cat	-:3(4)	A	A	A
Nasotrach	Unknown	-:3(4)	A	A	N
Wound	Unknown	A:3(4)	A	A	A
Pelvic abscess	Unknown	A:3(4)	A	A	A
Sputum	Unknown	A:3(4)	A	A	N
Liver abscess	Unknown	A:3(4)	A	A	N
Cat	Cat	-:3	A	A	A
Cat	Cat	-:3	A	A	A
Cat	Cat	-:3	A	N	A
Cat	Cat	D:3	A	A	A
Cat	Cat	-:3(1)	A	N	A
Cat	Cat	D:3(1)	A	A	A
Dog	Dog	-:3(1)	A	A	A
Dog	Dog	-:3(1)	A	A	A
Bite	Dog	-:4(3)	A	A	A
Blood	Unknown	A:4(3)	A	A	A
Eye	Unknown	A:4(3)	A	A	N
Sputum	Unknown	-:4(7)	A	A	A
Sputum	Unknown	A:4(12)	A	A	A
Spinal fluid	Unknown	-:5	A	A	A
Throat	Unknown	-:5	A	A	N
Bite	Unknown	-:5(1)	A	A	A
Bite	Cat	-:8(5)	A	A	A
Wound	Cat	D:8(13)	A	N	N
Dog	Dog	-:8	N	N	N
Bite	Dog	-:11	N	N	N
Human	Unknown	A:12(1,4)	A	A	N
Postoperative wound	Unknown	A:12(4,3)	A	A	A
Cat	Cat	-:12	A	A	A

Note: A - fermentation; N - no fermentation.

The type 3 cultures were isolated from wounds and internal infections, were associated with both cats and dogs, and included encapsulated and nonencapsulated cultures. Most of the somatic type 3(4) cultures had type A capsules. The fermentation reactions were a distinct feature of type 3 cultures. Most of the human type 3 cultures fermented the 3 sugars except for a few which did not ferment glycerol. Cultures from cats and dogs also had this pattern. Most of these cultures fermented the 3 sugars except for 2 of the cat cultures which did not ferment sorbitol. A type A:3(1,4) culture was isolated in Malaysia.

There were 5 type 4 cultures, 4 of which were from internal infections. Of those from internal infections, 3 had type A capsules. Four of the type 5 cultures fermented the 3 sugars and one fermented all but glycerol. None of the type 4 cultures was isolated from an animal but one was isolated from a dog bite.

There were 3 type 5 cultures; one was from a wound and 2 were from internal infections. None was associated with animals. None was encapsulated. Two cultures fermented the 3 sugars; one fermented all but glycerol. There were 3 type 8 cultures, 2 were isolated from cat bites and one from a dog culture. The type 8 cultures had variable fermentation reactions in the 3 sugars. One type 11 culture was isolated from a dog bite. As the dog cultures which were types 1 and 8, the type 11 culture did not ferment the 3 sugars. The 2 type 12 cultures isolated from humans cross-reacted with antisera to 2 other serotypes. One was from a postoperative wound and the other was from Malaysia. Both cultures had type A capsules and neither was associated with animals. One culture fermented the 3 sugars; one fermented

all but glycerol. One type 12 culture was isolated from a cat which fermented the 3 sugars.

In Table 11 the cultures are grouped according to their fermentation in mannitol, sorbitol and glycerol. The largest number of cultures fermented all 3 sugars. There were cultures of every somatic type except type 11 in this group. The type D cultures had this fermentation pattern, also. Twelve cultures were related to cats, 4 with dogs, and 20 with an unknown animal source.

The second largest group of cultures fermented mannitol and sorbitol but not glycerol. There were cultures representing all the somatic types except type 8 in this group. One culture was isolated from a dog bite, and 2 were from cats. Nine cultures were isolated from internal infections and 2 from bites.

One culture fermented mannitol and glycerol but not sorbitol. It was a type A:1 from the ear of a Vietmanese. Two cultures fermented mannitol but not sorbitol or glycerol. Both were isolated from infected cat bites.

Seven type 1 cultures, 1 type 8 and 1 type 11 did not ferment the 3 sugars. None of these cultures had type A capsules. Five of the cultures were from wounds; 1 was from an internal infection and 3 were from dogs.

In Table 12 are listed the serotypes of cultures isolated from cats and dogs, cat and dog bites and internal infections associated with cats and dogs. All the primary serotypes found in this study were associated with cats or dogs. None of these cultures was encapsulated.

Table 11. Cultures grouped by fermentation pattern.

Source	Serotype	Animal
Mannitol-A Sorbitol-A Glycerol-A		
Bronchial washing	-:1	Unknown
Sputum	-:1	Unknown
Bite	-:1	Unknown
Joint	-:1(4)	Unknown
Human	-:1(4)	Unknown
Sputum	A:1(5)	Unknown
Bite	-:1(7)	Cat
Cat	-:1	Cat
Bite	-:3	Dog
Bite	-:3	Cat
Abscess	-:3	Unknown
Sputum	-:3	Unknown
Spinal fluid and Blood	-:3	Dog
Cat	-:3	Cat
Cat	-:3	Cat
Cat	D:3	Cat
Bite	-:3(1)	Unknown
Wound	-:3(1)	Unknown
Human	-:3(1)	Unknown
Blood	A:3(1)	Unknown
Sputum	D:3(1)	Cat
Cat	D:3(1)	Cat
Dog	-:3(1)	Dog
Human	A:3(1,4)	Unknown
Bite	-:3(4)	Cat
Wound	A:3(4)	Unknown
Pelvic abscess	A:3(4)	Unknown
Bite	-:4(3)	Dog
Blood	A:4(3)	Unknown
Sputum	-:4(7)	Unknown
Sputum	A:4(12)	Unknown
Spinal fluid	-:5	Unknown
Bite	-:5(1)	Unknown
Bite	-:8(5)	Cat
Postoperative wound	-:12(4,3)	Unknown
Cat	-:12	Cat

continued

Table 11--continued

Source	Serotype	Animal
Mannitol-A Sorbitol-A Glycerol-N		
Blood	A:1(4)	Unknown
Bite	-:1(5)	Dog
Cat	-:1(5)	Cat
Cat	-:1(5)	Cat
Bronchial washing	-:3	Unknown
Sputum	A:3	Unknown
Blood	A:3	Unknown
Bite	-:3(1)	Unknown
Nasotrach	-:3(4)	Unknown
Sputum	A:3(4)	Unknown
Liver abscess	A:3(4)	Unknown
Eye	A:4(3)	Unknown
Throat	-:5	Unknown
Unknown	A:12(1,4)	Unknown
Mannitol-A Sorbitol-N Glycerol-A		
Ear	A:1	Unknown
Mannitol-A Sorbitol-N Glycerol-N		
Bite	-:1	Cat
Wound	D:8(13)	Cat
Mannitol-N Sorbitol-N Glycerol-N		
Wound	-:1	Dog
Bite	-:1	Dog
Bite	-:1	Cat
Pleural fluid	-:1	Unknown

continued

Table 11--continued

Source	Serotype	Animal
Mannitol-N Sorbitol-N Glycerol-N -- continued		
Bite	-:1(7)	Cat
Dog	-:1	Dog
Dog	-:1	Dog
Dog	-:8	Dog
Bite	-:11	Dog

Note: A - fermentation, N - no fermentation.

Table 12. Correlation of serotype with animal association.

Type	Number	Animal
-:1	4	2 dog bites, 2 dogs
-:1	3	2 cat bites, 1 cat
-:1(5)	3	dog bite, 2 cats
-:1(7)	1	dog bite
-:1(7)	1	cat bite
-:3	2	dog bite, spinal fluid and blood with dog association
-:3	4	cat bite, 3 cats
D:3	1	cat
-:3(1)	4	cat, 2 dogs, leopard bite
D:3(1)	2	cat, sputum with cat association
-:4(3)	1	dog bite
-:8	1	dog
-:8(5)	1	cat bite
D:8(13)	1	cat bite
-:11	1	dog bite
-:12	1	cat
Total	32	

In Table 13 the cultures are listed according to fermentation pattern and source. The sources are divided into the categories of cat or dog association or internal infections. Thirty-one of 55 cultures fermented the 3 sugars; 15 of these were from internal infections. Twelve cultures fermented mannitol and sorbitol but not glycerol; 9 of these were from internal infections. Nine cultures did not ferment the 3 sugars; 6 of these were associated with dogs, 2 with cats. Eleven of the 18 cat related cultures fermented the 3 sugars; 6 of the 11 dog related cultures were nonfermentative.

Table 13. Fermentation patterns and animal association.

Mannitol Sorbitol Glycerol	A A A	A A N	N N N	A N A	A N N	Total
Dog or dog bite	4	1	6	0	0	11
Cat or cat bite	12	2	2	0	2	18
Internal infection	15	9	1	1	0	26
Total	31	12	9	1	2	55

DISCUSSION

Some of the same somatic types of P. multocida were isolated from cats and dogs as were isolated from human infections. Somatic types 1, 3, and 12 were isolated from cats and types 1, 3 and 8 from dogs. Cultures of these same somatic types were also isolated from human infections. Specifically, cultures with somatic types 1, 3, 5 and 8 were isolated from wound infections, suggesting that cat or dog bites were the sources for these infections.

No type 4 cultures were isolated from cats or dogs. Most of the type 4 cultures were isolated from human internal infections. This would suggest a source other than cats or dogs for these infections. Most of the type 4 cultures of Blackburn et al. were recovered from poultry.[16] Other possible sources could be domestic and wild animals, human carriers,[72,130] human disease processes,[69] and soil.[93]

The results of this study were similar to those of Heddleston and Wessman,[61] and the compilation of Blackburn et al.[16] with one exception. Cultures in the above studies had primary somatic types 1, 3, 4, 6, 12, and 13 with types 3 and 4 predominating. Cultures of this study included the same primary somatic types except 6 and 13 with types 1 and 3 predominating. This disparity in the number of type 1 cultures may in part reflect the marked difference in the culture sources. In Heddleston and Wessman's study, 3 of the 30 cultures were from cat or dog bites while 16 out of 51 in this study were from animal bites.

In the same study, 1 type 1 culture was isolated from a laceration with an unknown source.[16] In this study, eight of the cultures from animal bites were type 1. In addition, type 1 cultures were isolated from 1 cat and 2 dogs. Heddleston and Wessman could have identified fewer type 1 cultures because there were so few bite cultures in their study.

Among the 30 cultures Heddleston and Wessman examined, there were 4 type 3 cultures which weakly reacted with type 4 antiserum and 4 type 4 cultures which weakly reacted with type 7.[61] The same reactions occurred in this study. Six type 3 cultures reacted weakly with type 4 and 1 type 4 reacted weakly with type 7. In addition, cultures with the following somatic types occurred, representing other secondary types: 1(4), 1(5), 1(7), 3(1), 3(1,4), 4(3), 4(12), 5(1), 8(5), 8(13), 12(4,1) and 12(4,3). Somatic types 1(5), 1(7), 3(1), 5(1), 8(5) and 8(13) were isolated primarily from animal bites. Again Heddleston and Wessman may not have identified cultures with these secondary reactions since they did not have many cultures from bite infections.

The data suggest that cultures with certain somatic types may be associated with particular animals. Types 1(5)and 12 were isolated from cats, type 8 from dogs and types 1, 3 and 3(1) from both cats and dogs. Type 3(4) cultures were not isolated from cats or dogs.

There appears to be correlation among the following colonial characteristics and the source of the culture: colony size and consistency on blood agar, optical variant and presence of a type A capsule. Cultures from dogs or dog bites were generally small, pasty, blue and nonencapsulated. Cat or cat bite cultures were usually medium, creamy, blue and nonencapsulated. Cultures from internal infections were either

like the 2 types above or large, mucoid, iridescent and possessing type A capsules. This agrees with prior observations of Smith[129] and Carter.[32]

Capsule production by 7 cultures was enhanced by passing them in mice. Five of these had been lyophilized and 2 had been subcultured on agar.

There appears to be a correlation between the presence of a type A capsule and the source of the culture. Many cultures isolated from internal infections had type A capsules while only one culture from a wound was type A. In many bacterial infections, encapsulated strains are more pathogenic than nonencapsulated ones. Thus, it would seem that the encapsulated P. multocida cultures cause some of the more severe internal infections. In addition, none of the cat and dog cultures had type A capsules. This confirms Smith's observation that cat and dog cultures did not have hyaluronic acid capsules. The above information also suggests a source, other than cats and dogs, for some internal infections.[128]

This study provides some more information on the fermentation reactions observed by Carter[32] and Smith.[129] Cultures with somatic type 1, 5, 8, 11 and 12 were generally consistent with prior findings, i.e., most cat cultures with these somatic types fermented mannitol, sorbitol and glycerol and most dog cultures did not. There were a few exceptions. There were 2 cat cultures which were somatic type 1 but did not ferment the 3 sugars. The type 3 cultures did not conform to these patterns, however. Most of the type 3 cultures from both cats and dogs fermented the 3 sugars. The fermentation patterns of the cultures from internal

infections seemed to be distinctive. Most of these cultures fermented mannitol and sorbitol but not glycerol, comprising the largest number of cultures having this pattern. In addition, another group of cultures from internal infections fermented the 3 sugars. This suggests that if these internal infections were secondary to bite infections, they were more often caused by cat than dog bites. Table 13 summarizes the data on fermentation reactions and culture source. It appears that most cat related cultures fermented the 3 sugars and that most dog related cultures did not. As Blackburn et al. found, there is not a complete correlation between fermentation pattern and animal source.[16] In addition, there did not seem to be a correlation between somatic type and fermentation pattern.

This is the first study to determine the capsular and somatic types of human cultures of P. multocida. It demonstrates that human cultures can be characterized in several useful ways. The somatic type can be determined by the system of Heddleston et al.[58] The human cultures in this study were the primary somatic types 1, 3, 4, 5, 8, 11, and 12. Some cultures reacted strongly with antiserum to one somatic type and less strongly with other antisera. To differentiate these cultures, the "secondary" somatic type should be written in parentheses following the primary somatic type. By doing this the somatic types found in this study were extended to types 1(4), 1(5), 1(7), 3(1), 3(4), 3(1,4), 4(3), 4(7), 5(1), 8(5), 8(13), 12(1,4) and 12(4,3). The presence of a capsule and its type can be determined. Type A capsules were often found on cultures from internal infections. Until a better system is developed, it is recommended that serotype identification

list first the capsular type followed by the somatic type with primary and secondary reactions when present. Thus a serotype might be designated A : 3(4). The fermentation reactions in mannitol, sorbitol and glycerol produce useful patterns and colonial characteristics on blood and dextrose starch agars may be distinctive.

The above characteristics, including the serotype along with the animal source could be used to characterize cultures of P. multocida. This kind of description could be useful in epidemiological studies. If a culture of P. multocida were isolated from a human infection and from an animal with which the human had had contact, and if the cultures were of the same serotype and possessed the same fermentation pattern, this information would strongly suggest that the animal was the source of the infection.

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