Corynebacterium renale No.115株有線毛菌感染マウスの腎臓,膀胱及び尿における有線毛菌から無線菌への集団変化

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Population Shift from Piliated to Non-piliated Bacteria in Kidneys, Bladder and Urine of Mice Infected with *Corynebacterium renale* Strain No. 115 Piliated Bacteria

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ABSTRACT. The shift of population from piliated (P^+) to non-piliated (P^-) bacteria was found in the kidneys, bladder and urine of the mice inoculated into the bladder with $10^{8.4}$ or more of *Corynebacterium renale* strain No. 115 P^+ bacteria. The results suggested that there was apparent selective *in vivo* growth of P^- bacteria, which were originally present small in number in the population of inoculated P^+ bacteria. The shift from P^+ to P^- bacteria was not found only in the mice with anti-pili antibodies response positive, nor was it accelerated by passive immunization of mice with anti-pili antiserum.—KEY WORDS: *Corynebacterium renale,* mouse, non-piliated, piliated, population shift.

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Non-piliated bacteria overgrew piliated bacteria in later stages of infection in a mouse model or rat model of human urinary infections with *Proteus mirabilis* [25], *Escherichia coli* [10], and *Klebsiella pneumoniae* [19], despite the fact that piliated bacteria had more infectivity than non-piliated bacteria did [7, 8, 16, 23]. In a model of hematogenous *P. mirabilis* pyelonephritis in rats, which bypassed the mucosal barrier, lightly piliated bacteria increased more than heavily piliated bacteria in the kidneys [25]. Apparent selection decreasing expression of type 1 pili of *E. coli* was found in the kidneys of mice infected with the mutant expressing P-fimbriae and type 1 pili [10]. In a model of retograde pyelonephritis in mice with *K. pneumoniae* piliated phenotype, it was found in the kidneys that non-piliated phenotype was the predominant population [19]. Lack of pili of freshly isolated human urinary pathogens was also reported [11, 20].

C. renale is the causal agent of bovine pyelonephritis and the first gram-positive bacteria which were reported to possess pili [28, 29]. Pili-mediated attachment of *C. renale* has been reported to tissue culture cells [14], mucous membrane of the urinary bladder [15] and epithelial cells of the bovine urinary bladder [27] and vulva [12]. But the role of pili in the pathogenesis of *C. renale* pyelonephritis is not known. No significant differences were found between the mice inoculated in the bladder with piliated (P^+) and non-piliated (P^-) bacteria of *C. renale* strain No. 115 in mortality, number of mice positive for recovery of the bacteria from the urine, bladder and kidneys and number of bacteria recovered from these urinary organs and tissues [9]. However, shift of population from P^+ to P^- bacteria was not examined in that study.

The shift of population from P^+ to P^- bacteria was found in the present study in the kidneys, bladder and urine of mice infected with *C. renale* strain No. 115 P^+ bacteria. The results are described below.

MATERIALS AND METHODS

Bacteria: P^+ clone of *C. renale* strain No. 115 was used. *C. renale* strain No. 115 was isolated in 1968 from the urine of an

apparently healthy cow raised in a herd where pyelonephritis occurred. P^+ clone of the strain was prepared previously [21] and has been maintained in the authors' laboratory. The clone was recloned by plating on nutrient agar before use in the present study as previously reported [9]. The proportion of piliated bacteria in the clone examined with 1000 colonies of the clone by the slide agglutination test with anti-pili antiserum was 99.9% for P^+ bacteria.

Medium and cultivation: Bacteria were cultivated on nutrient agar (pH7.2) at 37°C for 24 hr.

Antiserum: Rabbit antiserum against *C. renale* No. 115 P^+ or P^- bacteria was prepared as previously reported [13]. Rabbit anti-*C. renale* No. 115 P^+ bacteria antiserum was absorbed with the bacteria of *C. renale* strain No. 115 P^- clone [21]. The bacteria of P^- clone collected from the culture grown on nutrient agar were mixed with anti-P^+ clone serum at 37°C for 2 hr. The mixture was centrifuged and the supernatant was filtered through a membrane filter (Millipore Corp., 450 nm). The procedure was repeated until the absorption was completed, which was confirmed by the slide agglutination test using anti-pili and anti-P^- bacteria sera.

Mice: Female ddy-F mice (22–30g), 6 to 7 weeks old, were used. They were observed for a week to quarantine before use.

Methods of inoculation: Bacteria were suspended in phosphate-buffered saline (PBS, pH7.4) and inoculated into mice, as previously described [22]. The mice were anesthetized with ether and the abdominal wall incised. A bacterial suspension of 0.025 m*l* containing approximately $10^{7.7}$–$10^{9.5}$ colony forming units was inoculated into the urinary bladder and then the abdominal incision was closed.

Bacterial recovery: Mice were forced to urinate by pressing lightly the abdominal wall and 0.2 m*l* of serial dilution of the urine was inoculated on a nutrient agar plate daily. The kidneys and bladder were removed aseptically after the mice were sacrificed on the 7th post-infection day or after death of the mice until 7th post-infection day. The cross section of kidneys and the vertical section of bladder were stamped and spread on nutrient agar plates. In some experiments, the kidneys or bladder were homogenized in PBS. Homogenates of the kidneys or bladder were diluted serially, and 0.2 m*l* of the dilution was inoculated on a nutrient agar plate. The number of bacterial colonies recovered from 0.2 m*l* of serial dilution of kidneys, bladder homogenates or urine was counted.

Examination of recovered bacteria for piliation: The colonies grown on nutrient agar were examined for piliation by the slide agglutination test with anti-pili antiserum. When the colonies developed were small in number, less than 50, every colony was examined. When the colonies developed were larger in number, at least 50 colonies were examined. The colonies agglutinated with anti-pili antiserum were regarded as P^+ bacteria and those non-agglutinated were, P^- bacteria. The proportion (%) of P^- bacteria in each specimen was determined. The number of P^- or P^+ bacteria present in the urine was estimated from the proportion of P^- bacteria and the total number of recovered bacteria. Non-agglutinated colonies randomly selected were confirmed to be *C. renale* by the slide agglutination test with anti-P^- clone antiserum. Some colonies were examined for piliation by electron microscopy and the results agreed with those of the slide agglutination test.

Passive Immunization of mice with anti-pili antiserum: For passive immunization, mice were inoculated intraperitoneally with 0.1 m*l* of anti-pili antiserum 24 hr before inoculation with P^+ bacteria. The agglutination titer of the anti-pili antiserum was 1:2048.

Examination of anti-pili antibody in mice

Table 1. Proportion of P^- bacteria in the kidneys and baldder of the mice inoculated with P+ bacteria

No. of bacteria inoculated (log_{10})	Mouse No.	Termination	Proportion of P^- bacteria in		Anti-pili ELISA titer on 7th day
			Kidneys	Bladder	
9.5	1	7K[a]	% 36 (50)[c]	% 100 (50)[c]	<16
	2	4D[b]	16 (50)	16 (50)	NE
	3	7K	6 (50)	66 (50)	<16
	4	4D	6 (50)	14 (50)	NE
	5	7K	2 (50)	0 (50)	<16
	6	7K	NE	50 (50)	256
9.0	7	7K	100 (20)	66 (50)	<16
	8	7K	50 (8)	2 (50)	<16
	9	7K	26 (50)	40 (50)	128
	10	1D	6 (50)	2 (50)	NE
	11	1D	0 (2)	20 (20)	NE
	12	7K	NE	82 (50)	<16
	13	5D	0 (7)	0 (50)	NE
8.5	14	7K	24 (50)	22 (50)	<16
	15	4D	10 (50)	24 (50)	NE
	16	2D	8 (50)	NE[e]	NE
	17	7K	0 (50)	8 (50)	<16
	18	7K	0 (50)	0 (22)	<16
8.4	19	4D	60 (100)	38 (50)	NE
	20	3D	33 (3)	20 (100)	NE
	21	7K	1 (100)	0 (100)	128
	22	7K	0 (1)	0 (100)	<16
	23	7K	NE	0 (150)	256
7.7	24	3D	0 (230)	0 (100)	NE
	25–31	7K	0 (15–200)	0 (1–100)	32(1)[d], 64(1), 16(5)

a) Killed on 7th post-infection day.
b) Died on 4th post-infection day.
c) Number of colonies examined in parenthesis.
d) Number of mice examined for anti-pili ELISA titer in parenthesis.
e) Not examined.

sera by ELISA: A serum sample was obtained from each mouse at sacrifice. The serum antibody titer against pili was determined by ELISA using purified pili prepared as previously reported [9, 18]. ELISA titers 1:16 or less were considered negative [9].

RESULTS

Shift of population from P^+ to P^- bacteria in the kidneys and urinary bladder of the mice inoculated with P^+ bacteria. The bacteria recovered from the kidneys and bladder of the mice inoculated with P^+ bacteria were examined for piliation by the slide agglutination test with anti-pili antiserum. The results were shown in Table 1. The proportion of P^- bacteria in the kidneys and bladder apparently increased in the mice inoculated with $10^{8.4}$ or more of P^+ bacteria. The proportion of P^- bacteria in the kidneys varied from 0 to 100%, and was 10% or more in nearly half of the mice. The figures obviously show the increase of P^- bacteria in the kidneys, considering that the proportion of P^- bacteria in the inoculum was only 0.1%. All the 20 colonies recovered from the kidneys of mouse No. 7 were P^-. The proportion of P^- bacteria increased also in the bladder of the mice inoculated with $10^{8.4}$ or more of P^+ bacteria. Ten % or more of the colonies were P^- in more than half of the mice, and 50 to 100% of bacteria recovered from the bladder were non-piliated in 5 of 13 mice inoculated with $10^{9.0}$ or more of P^+ bacteria. On the contrary, P^- bacteria were never recovered from the kidneys and bladder of the mice inoculated with $10^{7.7}$ of P^+ bacteria.

Anti-pili antibody response was found by ELISA in 4 of 14 mice which were inoculated with $10^{8.4}$–$10^{9.5}$ P^+ bacteria and sacrificed on the 7th post-infection day. The titers of the anti-pili antibodies in the serum of mice ranged from 1:128 to 1:256 (Table 1).

As summarized in Table 2, the shift of population from P^+ to P^- bacteria in the kidneys was found, on the 7th post-infection day, in 2 of 2 (100%) mice with anti-pili antibody response negative. The shift of population in the bladder was found in 2 of 4 (50%) mice of anti-pili antibody response positive and 7 of 10 (70%) mice of anti-pili antibody response negative. The shift was negative or the proportion of P^- was low in mice Nos. 23 and 21 showing a high anti-pili ELISA titer. On the contrary, 100% shift was demonstrated in mice No. 1 (bladder) and No. 7 (kidneys), whose anti-pili ELISA titer was negative (Table 1). Thus, the shift of population from P^+ to P^- bacteria did not

occur only in the mice with anti-pili antibody response positive.

Shift of population from P^+ to P^- bacteria in the urine of mice inoculated into the bladder with $10^{8.4}$ of P^+ bacteria. Shift of population from P^+ to P^- bacteria in the urine was examined in 5 mice, Nos. 19–23 inoculated with $10^{8.4}$ of P^+ bacteria. Of these, 2 mice (Nos. 19 and 20) clearly demonstrated the shift of bacteria in the kidneys and bladder, as shown in Table 1. As shown in Fig. 1, the number of P^- bacteria in the urine, which was small on the 1st post-infection day (undetectable in mouse No. 19 and $10^{2.9}$ in mouse No. 20, in 0.02 m*l* of urine), immediately increased on the 2nd day to $10^{4.4}$ and $10^{4.5}$ in 0.02 m*l* of urine. The number of P^- bacteria then gradually decreased on the 3rd or 4th day, when the mice died. P^+ bacteria, on the other hand, were present in the urine at $10^{3.4}$ (mouse No. 19) and $10^{4.6}$ (mouse No. 20) in 0.02 m*l* of urine on the 1st post-infection day, increased slightly on the 2nd day and then gradually decreased, in parallel with P^- bacteria, on the 3rd or 4th day.

The proportion of P^- bacteria in the urine was 24, 40 and 18% on the 2nd, 3rd and 4th post-infection days in mouse No. 19, respectively, and 20 and 32% on the 2nd and 3rd post-infection days in mouse No. 20, respectively (data not shown but can be estimated from Fig. 1).

The results of the remaining 3 mice (Nos.

Table 2. Shift from P^+ to P^- bacteria in the kidneys and bladder and anti-pili antibody response on 7th day in the mice inoculated with $10^{8.4}$ or more of P^+ bacteria

Organ	Shift from P^+ to P^-	Anti-pili antibody response	
		+	−
Kidneys	+	2	6
	−	0	3
Bladder	+	2	7
	−	2	3

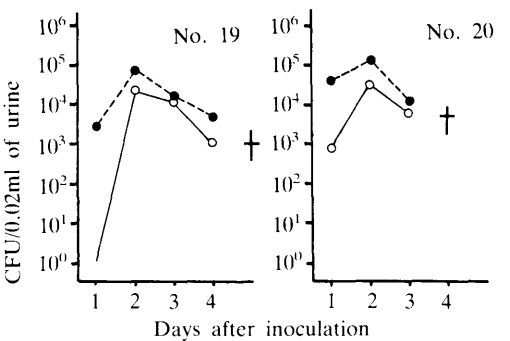

Fig. 1. The number of piliated and non-piliated bacteria recovered from the urine of mice (Nos. 19 and 20) inoculated into the bladder with $10^{8.4}$ of P^+ bacteria. Piliated bacteria: ●---● Non-piliated bacteria: ○—○ Mouse died: +.

Table 3. Comparison of shift of bacteria from P^+ to P^- in the kidneys between the mice passively immunized with anti-pili antiserum and those that were non-immunized

No. of bacteria inoculated (log_{10})	Anti-pili antiserum	No. of mice used	No. of mice in which shift from P^+ to P^- was found with the following proportion (%) of P^- bacteria in kidneys		
			0	2–50	>50
8.5	+	6	1	5 (2, 10, 20, 44, 50)[a]	0
	−	5	2	3 (8, 10, 24)	0
9.0	+	5	2	3 (8, 20, 21)	0
	−	6	2	3 (6, 26, 50)	1(100)

a) Percentage of P^- bacteria recovered from kidneys of each mouse in parenthesis.

21, 22 and 23), which showed only slight or no shift from P^+ to P^- bacteria in the kidneys and bladder, indicated that the proportion of P^- bacteria in the urine did not exceed 4% and 8% in 2 and 1 mouse, respectively.

Findings in the mice passively immunized with anti-pili antiserum. Eleven mice were passively immunized with anti-pili antiserum 24 hr before inoculation with $10^{8.5}$ or $10^{9.0}$ of P^+ bacteria, and the shift of bacteria from P^+ to P^- in the kidneys and bladder was examined. The passively immunized mice showed 1:64 to 1:256 agglutinin titer against P^+ bacteria prior to bacterial inoculation. The non-immunized 11 mice showed the agglutinin titer of less than 1:2.

Shift of bacteria from P^+ to P^- was found in the kidneys in both the passively immunized and the non-immunized mice. The proportion of P^- bacteria in the kidneys was essentially similar in the immunized and the non-immunized mice (Table 3). The proportion of P^- bacteria in the bladder was not different between the immunized and the non-immunized mice (data not shown).

DISCUSSION

The population shift from P^+ to P^- bacteria was found in the present study in the kidneys, bladder and urine of the mice inoculated into the bladder with $10^{8.4}$ or more of *C. renale* P^+ bacteria. The shift may be explained by selective *in vivo* growth of P^- bacteria, which were originally present in the population of inoculated P^+ bacteria at the rate of 0.1%.

Phyagocytosis may be a factor associated with the population shift of *C. renale*. Phagocytosis is believed to be the factor which makes non-piliated *P. mirabilis* [25], *E. coli*[10], or *K. pneumoniae* [19] advantageous in the kidneys of rats or mice compared with piliated bacteria. Piliated bacteria of these pathogens were more readily phagocytized and killed than P^- bacteria by phagocytic cells *in vitro* [2, 3, 4, 24, 26]. A recent *in vitro* study from the authors' laboratory showed that P^+ bacteria of *C. renale* strain No. 115, which were less phagocytized by mouse macrophage than P^- bacteria in the absence of opsonins, were phagocytized at significantly higher rate than P^- bacteria in the presence of anti-pili antiserum [17].

No apparent relationship was found in the present study between serum anti-pili antibodies and the shift of *C. renale* from P^+ to P^- bacteria; serum anti-pili antibodies which were found in the infected mice (Table 1 and 2) and in the mice passively immunized (intraperitoneally) with anti-pili antiserum 24 hr before inoculation of P^+ bacteria (Table 3) did not enhance the population shift. However, the influence of locally secreted anti-pili antibodies on the shift cannot be neglected. The anti-pili antibodies in the urinary tract of mice could not be examined in the present study. It was reported in rats that antibody-coated *C. renale* was present on the 2nd day of infection and that serum anti-*C. renale* antibody concentrations increased after antibody-coated bacteria appeared in the urine and kidneys [1]. The possibility that anti-pili antibodies were produced in the urinary tract of the mice inoculated with *C. renale* P^+ bacteria and that the antibodies exerted opsonic effects cannot be excluded. This point is a subject for future study.

P^- bacteria were recovered from the mice which were inoculated with $10^{8.4}$ or more of P^+ bacteria but not from the mice which were inoculated with $10^{7.7}$ of P^+ bacteria. Failure of detecting P^- bacteria in the mice inoculated with $10^{7.7}$ P^+ bacteria would be explained as follows. $10^{7.7}$ of P^+ bacteria contained $10^{4.7}$ of P^- bacteria as P^- bacteria were originally present in the population of inoculated P^+ bacteria at the rate of 0.1%. $10^{4.7}$ of P^- bacteria may disappear from the

urinary bladder without reaching the kidneys. It was reported that *C. renale* appeared to reach the kidneys in most of the mice inoculated into the bladder with 10^7 organisms, whereas the organisms disappeared from the urinary bladder without reaching the kidneys in many of the mice inoculated with less than 5×10^5 organisms [22].

Phase variation in type 1 pili of *E. coli* which occur in some cultural conditions [5, 6] was considered associated with population shift from piliated to non-piliated bacteria of *E. coli* and some other species of family Enterobacteriaceae *in vivo* [10, 19, 25]. Piliation of *C. renale* has been reported to be stable in various cultural conditions [29], being different from that of type 1 pili. Although phase variation has not been evidenced in pili of *C. renale,* its possibility and association with the population shift should be studied in the future.

Pili of *C. renale* isolated from diseased cows were reported to be small in number and not all of the bacterial population was piliated in the strains of *C. renale* [29]. The finding might be due to the shift of *C. renale* from P^+ to P^- bacteria in cows, as was reported in human urinary pathogens such as *E. coli* isolated from patients [11, 20].

Pili mediated bacterial adherence may be important at the first stage of *C. renale* infection. P^- bacteria of *C. renale,* however, may be advantageous compared with P^+ bacteria *in vivo,* as in the case of *E. coli* [10], *K. pneumoniae* [19] and *P. mirabilis* [25].

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要　約

Corynebacterium renale No. 115 株有線毛菌感染マウスの腎臓，膀胱及び尿における有線毛菌から無線菌への集団変化：福岡　隆，梁川　良（北海道大学獣医学部家畜衛生学教室）——*Corynebacterium renale* No.115 株の有線毛（P^+）菌を$10^{8.4}$またはそれ以上膀胱内に接種されたマウスの腎臓，膀胱及び尿において，P^+ 菌から無線毛（P^-）菌への集団変化が認められ，もともと接種 P^+ 菌の中に少数存在していた P^- 菌が生体内で選択的に増加したためと考えられた．P^+ 菌から P^- 菌への集団変化は，血中抗線毛抗体陰性のマウスでも認められ，わま，接種前にマウスを抗線毛抗体で受動免疫することによって促進されなかった．