INTRODUCTION

*Mycoplasma pneumoniae* is one of the smallest free-living bacteria, and one of the most common pathogens that cause upper and lower respiratory tract infection in humans, accounting for approximately 10–30% of all cases (1,2). *M. pneumoniae* infection could occur at any age. Its clinical presentations are various, including headache, sore throat, tracheobronchitis, and pneumonia. Furthermore, it is directly linked with a broad array of extra-pulmonary manifestations, sometimes life-threatening (3). *M. pneumoniae* spreads slowly and has an incubation time of approximately 23 days. Epidemics occur every 3–7 years and are usually 1–2 years in duration (4).

Analysis of the molecular characteristics of *M. pneumoniae* is essential for monitoring epidemic strains, for identifying the source of an outbreak, and for comparing the strain diversity within individual age groups. Currently, P1-restriction fragment length polymorphism analysis (RFLP), multi-locus variable number tandem repeat analysis (MLVA), multilocus sequence typing, single nucleotide polymorphisms, MALDI-TOF MS, and whole genome sequencing analysis have all been used for typing *M. pneumoniae* (5–8). However, P1 gene typing based on 2 repetitive elements (RepMP2/3 and RepMP4) in the gene MPN141 coding for P1 protein and MLVA typing based on the loci Mpn1, Mpn13, Mpn14, Mpn15, and Mpn16 are most commonly used.

P1 protein is considered as the major adhesin of *M. pneumoniae*. In the past 30 years, P1 gene typing has been the most common method to monitor *M. pneumoniae*. Kenri et al. in Japan reported a P1 genotype shift during different epidemic periods, type P1-1 and P1-2 showing alternative predominance in a population (9). The type-shift in dominant genotype from P1-1 to P1-2 was also found in our previous study (10). Type P1-1 and P1-2 strains in vitro formed biofilms that differ qualitatively and quantitatively, and P1-2 had a higher expression of community-acquired distress syndrome toxin that was a significant virulence factor specific to *M. pneumoniae* (11,12). Fan et al. found that *M. pneumoniae* pneumonia (MPP) patients infected with P1-1 strains exhibited a higher risk of developing severe MPP (13). However, Rodman et al. indicated that infections caused by P1-2 strains could be more severe than those caused by P1-1 strains (14). Multi-locus variable-number tandem-repeat analysis was first used to type *M. pneumoniae* in 2009, with the original protocol being amended many times since then. This typing method has a higher discriminatory power than P1 typing. Moreover, P1 types can be used for comparisons with previous studies, as a standard. However, no specific genotype has been identified to be associated with increased virulence or epidemic potential.

In general, due to the lack of a cell wall, a macrolide is chosen as the first-line antimicrobial agent for the treatment of *M. pneumoniae* infection, especially in children. However, reports of macrolide-resistant *M. pneumoniae* have appeared in Asia, Europe, and North America since the early 2000s (15–18). The
prevalence ranges from 1.00% in Slovenia to over 90.0% in Japan and China (19,20). Macrolide resistance of *M. pneumoniae* strains is mainly due to mutations in domain V of the 23S rRNA gene, with A2063G being the most prevalent mutation site, followed by A2064G (21). Studies focused on *M. pneumoniae* infection in children have been widely reported, but only limited studies have investigated the clinical and molecular characteristics of *M. pneumoniae* obtained from adults. In this study, we analyzed the P1 genotypes, MLVA types, amended MLVA types, and macrolide resistance associated gene mutations in *M. pneumoniae*-positive specimens, collected from children and adults during the same period, and directly compared the diversity of their molecular characteristics.

**MATERIALS AND METHODS**

*M. pneumoniae* specimens: In 2014–2015, 940 community-acquired pneumonia pediatric patients from the Affiliated Children’s Hospital of the Capital Institute of Pediatrics and 307 adult patients form Beijing Pu Ren Hospital were prospectively enrolled in a surveillance program. DNA samples were extracted using a QIAamp DNA minikit (Qiagen, Hilden, Germany). *M. pneumoniae* were confirmed using real-time polymerase chain reaction (PCR) as described previously. Reference strains FH (ATCC 15531) and M129 (ATCC 29342) were detected and typed as the positive control. Among the test samples, 271 (26.7%, 251/940) and 58 (18.9%, 58/307) were detected as *M. pneumoniae*-positive in children and adults, respectively. Due to the low DNA concentrations, only 247 (200 in children and 47 in adults) *M. pneumoniae*-positive DNA samples were successfully analyzed for their molecular characteristics. All experiments were carried out in compliance with the relevant laws and guidelines, in accordance with the ethical standards of the Declaration of Helsinki and were approved by the research board of the Ethics Committee of the Capital Institute of Paediatrics, in Beijing, China.

**PCR-RFLP typing of the P1 gene:** We used the nested PCR-RFLP performed as previously described to analyze the RepMP2/3 and RepMP4 (22).

*M. pneumoniae*-positive specimens were divided into P1 genotype 1 (P1-1) and P1 genotype 2 (P1-2). Then the variant types (2a, 2b, and 2c) of type P1-2 were determined. The most prevalent type, 2a, was determined in 1 specimen, and type 2c was identified in 4 adult specimens. Thus, the proportion of type P1-1 was not significantly higher in children than in adults (P > 0.05).

**Amended MLVA typing:** Five distinct amended MLVA types were identified among the 200 pediatric specimens, with M4-4-5-7-2, followed by M5-4-5-7-2 being the most prevalent types. Of the 47 adult specimens, 12 distinct MLVA types were identified. The most prevalent types were also M5-4-5-7-2, followed by M4-4-5-7-2. The proportions of type M4-4-5-7-2 and M5-4-5-7-2 did not differ significantly between children and adults (P > 0.05). Seven types - M2-4-5-7-3, M3-4-5-5-2, M4-4-5-6-2, M5-3-5-6-2, M6-4-5-6-2, M7-4-5-7-2, and M8-4-5-7-2 were identified only in pediatric specimens, whereas 4 types, M3-4-5-7-3, M4-4-5-7-3, M5-4-5-7-3, and M6-4-5-7-3, were identified only in adults (Table 1). The proportions of type M1-1 were not significantly higher in children than in adults (P > 0.05).

**RESULTS**

**P1 gene typing:** The 200 pediatric specimens were analyzed by RFLP. The results showed type P1-1 accounted for 89.5% (179/200) and type P1-2 accounted for 10.5% (21/200) (Table 1). In addition, variant type 2c was identified in 15 of the specimens typed as P1-2.

In the 47 adult specimens, the proportions of type P1-1 and type P1-2 were 87.5% (39/47) and 12.5% (6/47), respectively (Table 1). Among them, type 2a was determined in 1 specimen, and type 2c was identified in 4 adult specimens. Thus, the proportion of type P1-1 was not significantly higher in children than in adults (P > 0.05).

**MLVA typing:** Fifteen distinct MLVA types were identified among the 200 pediatric specimens, with M4-4-5-7-2, followed by M5-4-5-7-2 being the most prevalent types. Of the 47 adult specimens, 12 distinct MLVA types were identified. The most prevalent types were also M5-4-5-7-2, followed by M4-4-5-7-2. The proportions of type M4-4-5-7-2 and M5-4-5-7-2 did not differ significantly between children and adults (P > 0.05). Seven types - M2-4-5-7-3, M3-4-5-5-2, M4-4-5-6-2, M5-3-5-6-2, M6-4-5-6-2, M7-4-5-7-2, and M8-4-5-7-2 were identified only in pediatric specimens, whereas 4 types, M3-4-5-7-3, M4-4-5-7-3, M5-4-5-7-3, and M6-4-5-7-3, were identified only in adults (Table 1).

**Amended MLVA typing:** Five distinct amended MLVA types were identified among the 200 pediatric specimens: M3-5-6-2 (20/200, 10.0%), M4-5-5-2 (1/200, 0.5%), M4-5-6-2 (2/200, 1.0%), M4-5-7-2 (175/200, 87.5%), and M5-4-5-7-3 (2/200, 1.0%), with M4-5-7-2 being the most prevalent type. Three distinct MLVA types were found among the 47 adult specimens: M3-5-6-2 (8/47, 17.0%), M4-5-7-2 (30/47, 63.8%), and M4-5-7-3 (9/47, 19.1%), also with M4-5-7-2 being the most prevalent type. MLVA types M4-5-5-2 and M4-5-6-2 were identified only in pediatric specimens. The proportion of MLVA type M4-5-7-2 was significantly higher in children than in adults (P < 0.05) (Table 1). Overall, 99.4% (174/175) of pediatric specimens...
typed M4-5-7-2, 100% of specimens typed M4-5-5-2, M4-5-6-2, and M4-5-7-3 belonged to type P1-1, while 100% of specimens typed M3-5-6-2 and 0.57 (1/175) of specimens typed M4-5-7-2 belonged to type P1-2. In adult specimens, 100% of specimens typed M4-5-7-2, and M4-5-7-3 belonged to type P1-1, while 100% of specimens typed M3-5-6-2 belonged to type P1-2.

**Detection of macrolide resistance:** Macrolide resistance-associated gene mutations were detected in 90.5% (181/200) of the pediatric specimens and 76.6% (36/47) of the adults (Table 1). The mutation A2063G was determined in 178 pediatric specimens, A2064G

| Characteristic | Total no. (%) | No. collected in the children (%) | No. collected in the adults (%) |
|----------------|---------------|----------------------------------|--------------------------------|
| **MLVA(A) type** |               |                                  |                                |
| M2-3-5-6-2     | 5 (2.02)      | 3 (1.50)                         | 2 (4.26)                       |
| M2-4-5-7-2     | 21 (8.50)     | 18 (9.00)                        | 3 (6.38)                       |
| M2-4-5-7-3     | 2 (0.81)      | 2 (1.00)                         | –                              |
| M3-4-5-5-2     | 1 (0.40)      | 1 (0.50)                         | –                              |
| M3-4-5-7-2     | 36 (14.6)     | 31 (15.5)                        | 5 (10.6)                       |
| M3-4-5-7-3     | 3 (1.21)      | –                                | 3 (6.38)                       |
| M4-3-5-6-2     | 16 (6.48)     | 12 (6.00)                        | 4 (8.51)                       |
| M4-4-5-6-2     | 1 (0.40)      | 1 (0.50)                         | –                              |
| M4-4-5-7-2     | 66 (26.7)     | 57 (28.5)                        | 9 (19.1)                       |
| M4-4-5-7-3     | 2 (0.81)      | –                                | 2 (4.26)                       |
| M5-3-5-6-2     | 4 (1.62)      | 4 (2.00)                         | –                              |
| M5-4-5-7-2     | 57 (23.1)     | 48 (24.0)                        | 9 (19.1)                       |
| M5-4-5-7-3     | 3 (1.21)      | –                                | 3 (6.38)                       |
| M6-3-5-6-2     | 3 (1.21)      | 1 (0.50)                         | 2 (4.26)                       |
| M6-4-5-6-2     | 1 (0.40)      | 1 (0.50)                         | –                              |
| M6-4-5-7-2     | 21 (8.50)     | 17 (8.50)                        | 4 (8.51)                       |
| M6-4-5-7-3     | 1 (0.40)      | –                                | 1 (2.13)                       |
| M7-4-5-7-2     | 2 (0.81)      | 2 (1.00)                         | –                              |
| M8-4-5-7-2     | 2 (0.81)      | 2 (1.00)                         | –                              |
| **Amended MLVA type** |       |                                  |                                |
| M3-5-6-2       | 28 (11.3)     | 20 (10.0)                        | 8 (17.0)                       |
| M4-5-5-2       | 1 (0.40)      | 1 (0.50)                         | –                              |
| M4-5-6-2       | 2 (0.81)      | 2 (1.00)                         | –                              |
| M4-5-7-2       | 205 (83.0)    | 175 (87.5)                       | 30 (63.8)                      |
| M4-5-7-3       | 11 (4.45)     | 2 (1.00)                         | 9 (19.1)                       |
| **P1 genotype** |               |                                  |                                |
| Type 1         | 218 (88.3)    | 179 (89.5)                       | 39 (83.0)                      |
| Type 2         | 29 (11.7)     | 21 (10.5)                        | 8 (17.0)                       |
| **Macrolide resistant** |   |                                  |                                |
| Specimens     | 217 (87.9)    | 181 (90.5)                       | 36 (76.6)                      |
| **Macrolide susceptible** |   |                                  |                                |
| Specimens     | 30 (12.1)     | 19 (9.5)                         | 11 (23.4)                      |

1): A total of 247 specimens from the children and adults were analyzed.
2): A total of 200 specimens were obtained from the children.
3): A total of 47 specimens were obtained from the adults.
4): MLVA: multi-locus variable number tandem repeat analysis.
in 2 specimens, and A2063T in 1 specimen. Of the 47 adults specimens, 31 specimens harbored the mutation A2063G, and 3 specimens harbored A2064G, while A2063T and C2617T were found only in 1 specimen each.

Among the macrolide resistant pediatric specimens, 95.6% (173/181) were P1-1, while 4.42% (8/181) were P1-2. The composition of MLVA genotypes among the 181 macrolide-resistant specimens was: M4-5-7-2, 93.4% (169/181); M3-5-6-2, 3.87% (7/181); M4-5-6-2, 1.10% (2/181); M4-5-7-3, 1.10% (2/181); and M4-5-5-2, 0.55% (1/181). The rate of occurrence of the various mutant genotypes were: M4-5-7-2, 96.6% (169/175); M3-5-6-2, 35.0% (7/20); and M4-5-6-2, M4-5-7-3, M4-5-6-2, 100%. Among the macrolide resistant adult specimens, 91.7% (33/36) were P1-1, while 8.33% (3/36) were P1-2. The composition of MLVA genotypes among the 36 macrolide-resistant specimens was: M4-5-7-2, 72.2% (26/36); M3-5-6-2, 8.33% (3/36); and M4-5-7-3, 19.4% (7/36). The rate of occurrence of the various mutant genotypes were: M4-5-7-2, 86.7% (26/30); M3-5-6-2, 37.5% (3/8); and M4-5-7-3, 77.8% (7/9).

Although the proportions of P1 genotypes were similar among resistant specimens, the rate of occurrence of mutant genotype M4-5-7-2 was significantly higher than that of genotype M3-5-6-2 (P < 0.05) in both age groups.

DISCUSSION

*M. pneumoniae* infections are more common in children than in adults. Limited studies have investigated *M. pneumoniae* obtained from adults, and compared it with isolates from children (27–30).

In the present study, type P1-1 was the predominant genotype in both children and adults; this finding was in agreement with other studies in China and the United States (26,27). Although the distribution of P1 genotype showed no statistical difference between children and adults in China, the proportion of P1 genotype in children (84.4%) was significantly higher than the proportion in adults (64.3%) in the United States (28). Obviously, there was a regional difference in P1 genotype distribution.

Fifteen and 12 distinct MLVA types were identified in child and adult specimens, respectively. M4-4-5-7-2 and M5-4-5-7-2 were the most common types in both age groups. This was slightly different from what was reported by Qu et al. in 2010–2012, which might result from period differences (27).

By using the amended MLVA typing method using 4 loci, pediatric specimens were classified as 5 MLVA types with M4-5-7-2 and M3-5-6-2 as the prevalent ones, while adult specimens were divided into only 3 types with M4-5-7-2 as the most prevalent. However, the proportion of M4-5-7-2 in children (87.5%) was significantly higher than the proportion in adults (P < 0.05) (27). The distribution of MLVA types in adults also paralleled previous study in China (63.8% VS 75.7%) (P > 0.05) (27). There, MLVA types in children were more diverse, and type M4-5-5-2 and M4-5-6-2 were only reported in pediatric specimens. In the present study, we also found nearly all of the specimens typed M4-n-n-n were P1-1, while the specimens typed M3-n-n-n were P1-2 in children and adults.

Macrolide resistance in *M. pneumoniae* (MRMP) strains highly correlates with longer duration of fever and hospitalization, more serious radiological findings, and more extrapolmonary complications (29). The resistance rates to macrolides in both age groups in our study were extremely high. The rate in children (91.0%) was significantly higher than the rate in adults (76.6%), so MRMP might be particularly severe in children. The point mutations A2063G, A2063T, A2064G, and C2617T were detected in the present study. Among them, 97.8% in pediatric MRMP specimens, compared with 86.1% in adults, harbored the A2063G mutation.

Macrolide resistance is usually considered to correlate with the widespread use of macrolides for respiratory tract infections in China. However, in the present study, the rate of occurrence of mutations of the most prevalent type M4-5-7-2 was 96.6% in children, while it was 72.2% in adults, and the difference was statistically significant. The resistance rate of genotype M4-5-7-2 was significantly higher than that of genotype M3-5-6-2 (P < 0.05) in both age groups. Other studies in Beijing and Hong Kong also showed that there was a possible association between M4-5-7-2 and macrolide resistance (10,27,30). In our previous study, we also indicated that there was a decline in macrolide resistance rate in 2014–2015 compared with that in 2008–2013, and this trend followed a slight type shift in M4-5-7-2 and M3-5-6-2 (10).

There were 2 limitations in our study. Firstly, the clinical features of patients infected with *M. pneumoniae* have not been analyzed. Another limitation is that the number of *M. pneumoniae*-positive specimens in adults was small. In China, besides the low positive detection rate, *M. pneumoniae* infection in adults would not attract as much attention as the infection in children. In the initial stage, most adults choose to take antibiotics by themselves. Therefore, we can collect few adult *M. pneumoniae* specimens. Analysis of more adult specimens will help to better understand the differences.

In conclusion, P1 genotype 1 and MLVA type M4-5-7-2 were predominant in both age groups in Beijing. We also observed differences in distribution of *M. pneumoniae* genotypes between children and adults. Otherwise, *M. pneumoniae* had a higher rate of macrolide resistance in children and macrolide resistance was correlated with MLVA type M4-5-7-2. Although more diverse genotypes and a higher prevalence of macrolide resistance were found in the pediatric specimens, our results were not enough to define any specific genotype responsible for the high prevalence of *M. pneumoniae* infection in children. Further investigations are warranted to help to better understand the difference of molecular characteristics and clinical severity in different age groups.

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Conflict of interest  None to declare.
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