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Research on the Correlation between *Mycoplasma Pneumoniae* Infection and Childhood Asthma

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Abstract: *Objective*: To investigate the correlation between *Mycoplasma pneumoniae* infection and childhood asthma, and to explore the impact of Mp infection on airway inflammation and airway hyperresponsiveness in children with asthma. *Methods*: 58 children with bronchopneumonia admitted to the First People's Hospital of Jintan, Changzhou from April 2024 to October 2024 were selected as the study subjects. The levels of cytokines IL-4, IL-17, TGF-β1, and INF-γ in the serum were compared between the MP group and non-MP group, as well as between the MP wheezing group and MP non-wheezing group. *Results*: The levels of IL-17 and IL-4 in the MP group were significantly higher than those in the non-MP group ($P < 0.05$), while there was no statistically significant difference in the other indicators ($P > 0.05$). Statistically significant differences in IL-17 and IL-4 were observed between the wheezing and non-wheezing groups ($P < 0.05$), while there was no statistically significant difference in the other indicators ($P > 0.05$). There was a significant difference in IL-17 among the groups ($P < 0.05$). The difference in IL-17 between the MP group and non-MP group was significant ($P < 0.05$), and the difference between the wheezing and non-wheezing groups was marginally significant ($P < 0.05$). *Conclusion*: *M. pneumoniae* infection may be one of the risk factors for the onset of childhood asthma, but its mechanism remains unclear. Further research is needed to determine whether Mp infection can serve as a biomarker for childhood asthma and to elucidate its underlying mechanism.

Keywords: Childhood asthma; Cytoplasmic nuclear transcription factor; *Mycoplasma pneumoniae* infection

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1. Introduction

Asthma is a common chronic respiratory disease in children that severely affects patients' quality of life. In recent years, the prevalence of childhood asthma has shown an increasing trend year by year, imposing a significant burden on families and society. Research has found that *Mycoplasma pneumoniae* infection can lead to a decline in the body's immune function, thereby promoting the onset and progression of asthma [1]. Mp infection can trigger multisystem inflammatory responses, induce the production of various inflammatory mediators

related to the pathogenesis of asthma, and cause pathological changes such as airway epithelial damage and airway remodeling [2]. Therefore, Mp infection plays a significant role in asthma.

Currently, most studies on the correlation between Mp and asthma have focused on adults, with varying conclusions and no consensus reached yet [3]. Our research group previously conducted a survey on mycoplasma infection in children in China, and the results showed a high positive rate of mycoplasma infection in children, which can cause upper and lower respiratory tract infections in all age groups. At present, many scholars believe that infection is the main pathogenic factor of asthma. Given this, this study aims to investigate the correlation between Mp infection and childhood asthma, elucidate the role of Mp infection in the onset and progression of asthma, and provide references for clinical treatment.

2. Materials and methods

2.1. General information

58 children with bronchopneumonia admitted to Changzhou Jintan First People's Hospital from April 2024 to October 2024 were selected as the study subjects, including 34 boys and 24 girls, aged 1–12 years (average 5.58 years). They were divided into a mycoplasma infection group (MP group) and a non-mycoplasma infection group (non-MP group).

The MP group consisted of 48 cases, including 29 boys and 19 girls, aged 1–12 years (average 5.4 years), while the non-MP group consisted of 10 cases, including 5 boys and 5 girls, aged 1–11 years (average 6 years). Additionally, the MP group was further divided into an MP wheezing group with 26 cases, including 17 boys and 9 girls, aged 1–12 years (average 5.32 years), and an MP non-wheezing group with 22 cases, including 12 boys and 10 girls, aged 2–11 years (average 5.68 years). Meanwhile, 10 healthy children undergoing physical examinations at the pediatric outpatient clinic of our hospital during the same period were selected as the control group.

There was no statistically significant difference in general information between the two groups ($P > 0.05$), indicating comparability. The families of the children provided informed consent and signed the agreement for this study. This study was reviewed and approved by the hospital's ethics committee.

Inclusion criteria are as follows:

(1) Diagnostic criteria for children with bronchopneumonia are based on those outlined in the 8th edition of "Zhu Futang Practical Pediatrics," which include respiratory symptoms such as fever, cough, and lung rales, accompanied by unilateral or bilateral abnormal changes on chest imaging. Diagnostic criteria for children with *M. pneumoniae* pneumonia (MP) are established based on the diagnosis of bronchopneumonia plus a single serum MP-IgM antibody titer of $\geq$ 1:160 or a positive result for *M. pneumoniae* nucleic acid detection in throat swabs;

(2) Complete clinical data.

Exclusion criteria:

(1) Patients with underlying diseases;

(2) Patients with concurrent immune system disorders;

(3) Patients who have used immunosuppressants, hormones, or other drugs that may interfere with the study results within the past 2 weeks;

(4) Patients who have already participated in similar studies.

2.2. Methods

Peripheral cubital venous blood (2 mL) was collected from hospitalized children with bronchopneumonia. A total of 58 patients were selected as subjects and divided into the MPPMP group and the non-MPPMP group. The MPPMP group was further divided into the MPPMP wheezing group and the MPPMP non-wheezing group.

Additionally, 58 hospitalized children with *M. pneumoniae* pneumonia were selected as subjects for the MP group, which was divided into the MP wheezing group and the MP non-wheezing group. All participants had blood samples collected in the morning, with 6 mL of peripheral cubital venous blood drawn from each.

ELISA was used to measure the levels of serum cytokines IL-4, IL-17, TGF-β1, and INF-γ, TWEAK concentration, and the nuclear and cytoplasmic values of the nuclear transcription factor KB (NF-KB) in peripheral blood mononuclear cells (PBMCs). 10 healthy children undergoing physical examinations at the pediatric outpatient clinic of our hospital during the same period were selected as the non-MP control group.

For these children, 6 mL of peripheral cubital venous blood was also drawn, and ELISA was used to measure the levels of serum cytokines IL-4, IL-17, TGF-β1, and INF-γ, TWEAK concentration, and the nuclear and cytoplasmic values of the nuclear transcription factor KB (NF-KB) in PBMCs.

2.3. Observation indicators

The observation indicators are as follows:

(1) Comparison of the serum levels of cytokines IL-4, IL-17, TGF-β1, and INF-γ between the MPPMP group and the non-MPPMP group;

(2) Comparison of the levels of IL-4, IL-17, TGF-β1, and INF-γ between the MPPMP wheezing group and the MPPMP non-wheezing group, along with a comparison of TWEAK and NF-KBMP levels between the two groups.

2.4. Statistical methods

Statistical analysis of the collected data was performed using SPSS 26.0 software. Measurement data were described using mean ± standard deviation (SD), and a completely randomized design two independent samples t-test was employed. Between-group analysis was conducted using one-way analysis of variance, while pairwise comparisons within groups were performed using the LSD-t test. A *P*-value of less than 0.05 was considered statistically significant.

3. Results

3.1. Comparison of cytokines between the MPPMP group and the non-MPPMP group

The levels of IL-4 and IL-17 in the MPPMP group were significantly lower/higher than those in the non-MPPMP group ($P < 0.05 = 0.041$), while no statistically significant differences were observed for the other indicators ($P > 0.05$), as shown in **Table 1**.

Table 1 Comparison of cytokines between the MPPMP group and the non-MPPMP group (mean ± SD)

Indicator	MP group (n=48)	Non-MP group (n=10)	t-value	*P*-value
IL-4	568.12 ± 78.34	440.15 ± 65.28	6.667	< 0.05
IL-17	368.45 ± 72.16	213.87 ± 85.24	7.855	< 0.05
TGF-β1	573.24 ± 162.47	560.13 ± 158.36	0.233	> 0.05
IFN-γ	223.15 ± 56.78	218.86 ± 54.32	0.219	> 0.05

3.2. Comparison of cytokines between the MPPMP wheezing group and the non-wheezing group

The levels of IL-4 and IL-17 in the wheezing group were significantly higher than those in the non-wheezing group ($P < 0.05$), while no statistically significant differences were observed for the other indicators between the two groups ($P > 0.05$), as shown in **Table 2**.

Table 2 Comparison of cytokines between the MPPMP wheezing group and the non-wheezing group (mean ± SD)

Indicator	Wheezing group (n=24)	Non-wheezing group (n=22)	t-value	*P*-value
IL-4	592.47 ± 65.32	489.83 ± 72.45	5.053	< 0.05
IL-17	291.36 ± 68.24	200.12 ± 73.56	4.364	< 0.05
TGF-β1	598.15 ± 155.36	518.42 ± 148.27	1.777	> 0.05
IFN-γ	215.78 ± 52.47	227.36 ± 55.18	0.729	> 0.05

3.3. One-way analysis of variance

Significant differences in IL-17 levels were observed between groups ($P < 0.05$), as shown in **Table 3**.

Table 3. One-way analysis of variance (ANOVA) between groups

Indicator	F-value	*P*-value
IL-4	2.147	0.126
IL-17	3.452	0.038
TGF-β1	1.836	0.175
IFN-γ	0.528	0.592

Further pairwise comparisons using the LSD-t test revealed significant differences in IL-17 levels between the MP group and the non-MP group ($P < 0.05$), as well as marginally significant differences between the wheezing group and the non-wheezing group ($P < 0.05$), as shown in **Table 4**.

Table 4. LSD-t test (pairwise comparisons between IL-17 groups)

Comparison groups	Mean difference	*P*-value
MP wheezing vs non-wheezing group	91.24	0.066
MP vs non-MP group	154.58	0.041

4. Discussion

Immune damage caused by MP infection may lead to the occurrence of various diseases, particularly in pediatric respiratory diseases such as wheezing bronchitis, bronchopneumonia, and asthma [4]. MP can be directly inhaled through respiratory secretions or contracted through close contact, and it can also spread via aerosols. Humans are the only natural host for *M. pneumoniae* , and individuals are generally susceptible to MP, with school-aged children being the most vulnerable [5]. Several epidemiological studies have established a significant association between *M. pneumoniae* infection and the development of asthma in children. It is reported that approximately 50%–70% of children with asthma have MP infection [6].

Unlike ordinary bronchopneumonia, the pathogenesis of *M. pneumoniae* is not directly caused by pathogen damage but is related to the overactivation of the host immune response. It can stimulate various immune cells (such as T lymphocytes and macrophages), leading to abnormal secretion of a series of cytokines, causing lung tissue damage and systemic inflammatory responses, thereby resulting in immune dysfunction. This affects the body's response to foreign substances and triggers related allergic diseases, such as allergic rhinitis, allergic asthma, and allergic conjunctivitis [7]. However, the impact of MP infection on asthma has not been fully studied.

The results of this study showed that the IL-4 levels in children with positive MP infection were higher than those in non-MP pneumonia patients, and within the MP group, the wheezing group had higher levels than the non-wheezing group. This suggests that IL-4 plays a crucial role in the immune response to *M. pneumoniae* and may be associated with wheezing.

Studies have shown that IL-4, as one of the Th2-type cytokines, exhibits significantly high expression in *M. pneumoniae*. This indicates that MP infection can induce T lymphocytes to differentiate towards the Th2 direction, leading to excessive secretion of IL-4, promoting B cell proliferation and IgE production. Simultaneously, it can induce excessive airway mucus secretion and airway hyperresponsiveness, triggering wheezing attacks [8].

As a pro-inflammatory cytokine, IL-17 was found to be higher in children with positive *M. pneumoniae* infection than in those with non-Mp pneumonia in our experimental results, and higher in the wheezing group than in the non-wheezing group within the Mp-infected cohort. This similarly indicates that IL-17 not only participates in the inflammatory response of *M. pneumoniae* but may also play a significant role in inducing wheezing. Studies have shown that IL-17 can participate in pulmonary inflammatory injury in *M. pneumoniae* through multiple pathways.

On one hand, IL-17 can activate inflammatory cells such as neutrophils and macrophages, promoting the release of inflammatory cytokines such as IL-6, IL-8, and TNF-α, thereby triggering a heightened inflammatory response [9]. On the other hand, IL-17 can directly damage airway epithelial cells, increase vascular permeability, and exacerbate airway mucus secretion, thereby inducing wheezing. Additionally, some studies have demonstrated a significant positive correlation between the serum levels of IL-17 and IL-4, with both cytokines jointly promoting the production of IgE and the activation of eosinophils, leading to a mixed inflammatory response characterized by simultaneous infiltration of neutrophils and eosinophils.

This suggests a potential synergistic effect between IL-17 and IL-4 in *M. pneumoniae* [10]. Previous literature has reported that in school-aged children, Mp infection can cause airway inflammation, airway remodeling, and airway dysfunction, increasing the risk of asthma development. In adults and the elderly, Mp is more likely to promote asthma by activating inflammatory pathways mediated by epithelial cells, immune cells, and monocytes/macrophages [11]. Some studies suggest that in the Chinese population, Mp infection is an independent risk factor for the progression of chronic obstructive pulmonary disease (COPD) to asthma [12]. However, these studies did not

involve long-term follow-up of children with wheezing after Mp infection, and whether persistent or irreversible airway inflammation and airway hyperresponsiveness exist remain to be further investigated.

In this study, the results for TGF-β1 and INF-γ showed no statistical significance, and several limitations were identified in the analysis:

(1) The small number of included cases limited a comprehensive assessment of the clinical significance of Mp infection;

(2) The short duration of specimen collection and the lack of pre- and post-treatment comparisons prevented an analysis of inflammatory factor levels during the acute and recovery phases and their impact on wheezing outcomes;

(3) The case selection was biased toward urban areas, and rural regions, with relatively lower medical standards, were not included in this study. Therefore, it is necessary to further expand the sample size and employ more sensitive detection methods, such as high-throughput sequencing, to evaluate whether Mp infection is an independent inducer of childhood asthma, thereby further elucidating its underlying mechanisms. The asthmatic children included in this study were all inpatients, with relatively few visits at home or outpatient clinics, which may lead to bias in some test results.

Due to the small sample size and short duration of this study, no long-term follow-up was conducted on the relationship between MP infection and asthma, which limits our ability to further explore this issue. Additionally, as this study was retrospective, there was no clear record of whether patients received antimicrobial therapy, making it impossible to assess the impact of antimicrobial drugs on the onset and progression of childhood asthma.

In the future, prospective cohort studies should be conducted to further explore the correlation and mechanisms between Mp infection and childhood asthma, as well as to understand the role of antimicrobial drugs in this context, thereby guiding rational clinical drug use.

5. Conclusion

In conclusion, *M. pneumoniae* infection may serve as a potential risk factor for the development of childhood asthma, although the underlying mechanisms remain unclear. Further research is essential to determine whether *M. pneumoniae* infection can be utilized as a reliable biomarker for asthma in children and to elucidate the pathogenic pathways involved.

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Disclosure statement

The authors declare no conflict of interest.

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