Natural Resources for Human Health
eISSN: 2583-1194

Original Research

Received 09 July 2026
Revised 20 Aug 2026
Accepted 07 Sep 2026

Natural Resources for Human Health 2026, Vol. 6 (20s): 31–36

KEYWORDS:
community-acquired pneumonia; childhood overweight; intestinal microbiota; respiratory microbiota; dysbiosis; gut–lung axis.

Gut And Respiratory Microbiota in Overweight Children with Community-Acquired Pneumonia: A Comparative Study

Ch. L. Davronova[1], N. D. Azizova[2], F. M. Shamsiev[2], Sh. B. Uzakova[2]

[1]Termez Branch of Tashkent Medical Academy, Termez, Uzbekistan.
[2]Republican Specialized Scientific-Practical Medical Center of Pediatrics, Ministry of Health of the Republic of Uzbekistan, Tashkent, Uzbekistan.
Corresponding author: Ch. L. DAVRONOVA. Email: ch.davronova@yahoo.com

ABSTRACT: Altered microbial communities may modify respiratory inflammation in children with excess body weight. This prospective comparative study examined culture-based intestinal and upper-airway microbiota findings and clinical characteristics in 60 hospitalized children aged 3–7 years with radiologically confirmed community-acquired pneumonia (CAP): 30 with overweight or obesity and 30 with normal weight; 20 healthy children were also enrolled as controls. Nasopharyngeal, oropharyngeal and stool specimens were collected, and systemic inflammatory markers were assessed. Compared with normal-weight patients, overweight/obese children had more bronchial obstruction (63.3% vs 30.0%), prolonged cough (73.3% vs 43.3%) and elevated C-reactive protein (32.4 ± 6.9 vs 20.8 ± 4.7 mg/L; source-reported summary values). Reduced Bifidobacterium spp. was recorded in 76.7% versus 30.0%, and grade III dysbiosis in 46.7% versus 13.3%. Upper-airway mixed microbial growth was observed in 53.3% versus 23.3%. The source reported an inverse association between Bifidobacterium counts and C-reactive protein ($r = -0.61$) and a positive association between dysbiosis grade and length of stay ($r = 0.58$). These observations suggest associations among nutritional status, culture-defined microbial imbalance and the clinical course of CAP. Because the intervention protocol and several analytic details were not supplied, a specific treatment benefit or causal gut–lung mechanism cannot be established.

1. INTRODUCTION

Community-acquired pneumonia remains a major contributor to childhood morbidity and hospital use. Its severity reflects interactions among the infectious agent, host immune responses, nutritional status and clinical management. International guidance emphasizes careful assessment and evidence-based treatment rather than assuming a single cause of respiratory symptoms (World Health Organization, 2024; Bradley et al., 2011).

Excess adiposity is associated with metabolic and inflammatory changes that can influence susceptibility to infection and host responses. The intestinal microbiota interacts with metabolic regulation and both innate and adaptive immunity; disruption of these interactions has been described in metabolic disorders (Fan and Pedersen, 2021; Zheng et al., 2020). However, such mechanistic insights do not establish the direction of causality in an individual clinical study.

The gut–lung axis is a useful conceptual framework linking microbial communities, microbial metabolites, mucosal barriers and respiratory immune signaling. Evidence from experimental and observational work indicates that microbiota-derived signals may influence airway immunity, but their contribution to acute childhood pneumonia requires clinically rigorous investigation (Dang and Marsland, 2019; Özçam and Lynch, 2024).

The present study addresses the following question: are excess body weight and culture-defined intestinal and upper-respiratory microbial disturbances associated with greater clinical and inflammatory burden among preschool children hospitalized with CAP? A secondary exploratory aim was to describe the reported within-group clinical changes during comprehensive treatment, without making an unsupported causal claim about an incompletely characterized microbiota-directed component.

2. MATERIALS AND METHODS

2.1 Study design and participants

According to the source manuscript, this was a prospective comparative study including 60 children aged 3–7 years hospitalized with clinically and radiologically confirmed CAP. Group 1 comprised 30 children classified as overweight or obese, and group 2 comprised 30 normal-weight children. An additional 20 apparently healthy children of comparable age formed a control group. Reported inclusion conditions were a confirmed CAP diagnosis, age 3–7 years and hospitalization within 72 hours of disease onset. The source provided no numerical control-group findings, so the present results focus on comparisons between the two CAP groups.

2.2 Clinical and microbiological assessments

Anthropometric findings and routine clinical and laboratory results were recorded. Fever, bronchial obstruction, duration of cough, dyspnea, prior pneumonia, peripheral leukocyte count, erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), duration of hyperthermia and hospitalization duration were compared. Nasopharyngeal and oropharyngeal swabs were reportedly taken before initiation of antibacterial treatment, and stool specimens were submitted for quantitative culture of selected commensal and opportunistic organisms. The source describes culture-based assessments rather than sequencing-based microbiome profiling.

The original methods mention interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α) and interleukin-10 (IL-10), but provide no assay characteristics or numerical results for these biomarkers; therefore, no cytokine values or between-group effects are reported here. Positive upper-airway cultures are interpreted as organism detection or colonization and should not, on their own, be treated as proof of causative pneumonia pathogens.

2.3 Treatment and outcome interpretation

The source describes usual pneumonia management combined with unspecified microbiota-directed correction in the overweight/obesity group. It reports selected measurements before and after treatment (Table 5) but does not identify the microbiota product or regimen, allocation method, timing of observations, concomitant antibiotic regimen, adverse-event monitoring or a comparable pre/post treatment table for the normal-weight group. Consequently, treatment-associated observations are presented descriptively and cannot determine the independent efficacy of microbiota correction.

2.4 Statistical analysis

The source identifies IBM SPSS Statistics and a nominal significance threshold of $p < 0.05$, but does not specify the statistical tests for individual comparisons, distributional assumptions, the meaning of $M \pm m$, or adjustment for multiple comparisons. For transparency, the p values in Tables 1, 3 and 4 below were recalculated from the reported 2×2 aggregated counts with two-sided Fisher exact tests ($n = 30$ per CAP group); these exploratory recalculations replace incompatible thresholds printed in the source. Continuous-variable means and dispersions, and the two correlation coefficients, are retained exactly as reported in the source; their significance cannot be independently verified without participant-level data. No raw participant dataset was available.

3. RESULTS AND DISCUSSION

3.1 Clinical presentation

Children with excess body weight had more frequent bronchial obstruction and prolonged cough than the normal-weight CAP comparison group (Table 1). The differences in fever, dyspnea and a history of recurrent CAP were directionally similar, but did not meet the 0.05 threshold when the published counts were evaluated using two-sided Fisher exact tests. These descriptive group differences may reflect variations in underlying respiratory or metabolic vulnerability, although the study does not provide covariates for adjustment.

Table 1. Clinical features of community-acquired pneumonia by weight group

Clinical characteristic	Overweight/obese (n = 30)	Normal weight (n = 30)	Fisher p
Fever > 38.5 °C	24 (80.0%)	16 (53.3%)	0.054
Bronchial obstructive syndrome	19 (63.3%)	9 (30.0%)	0.019
Prolonged cough (> 10 days)	22 (73.3%)	13 (43.3%)	0.035
Dyspnea	15 (50.0%)	7 (23.3%)	0.060
Previous episodes of CAP	11 (36.7%)	4 (13.3%)	0.072

Note. Two-sided Fisher exact tests were calculated from the reported group counts. These p values may differ from those in the original source; no multiplicity correction was applied.

3.2 Systemic inflammation and duration of illness

Compared with their normal-weight peers, the overweight/obese CAP group had higher source-reported leukocyte count, ESR and CRP, as well as longer episodes of fever and hospital stays (Table 2). CRP was reported as 32.4 ± 6.9 mg/L versus 20.8 ± 4.7 mg/L. The source labels continuous variables M ± m; however, it does not define m or supply individual observations, and therefore no independent mean-comparison p values can be stated. These differences warrant confirmatory analysis adjusted for age, sex, obesity classification, severity at presentation and treatment exposures.

Table 2. Source-reported inflammatory markers and clinical duration

Outcome	Overweight/obese (n = 30)	Normal weight (n = 30)
Leukocytes ($\times 10^9$/L)	13.2 ± 1.9	10.7 ± 1.5
ESR (mm/h)	27.1 ± 4.8	18.6 ± 3.9
C-reactive protein (mg/L)	32.4 ± 6.9	20.8 ± 4.7
Duration of hyperthermia (days)	5.0 ± 0.9	3.3 ± 0.8
Length of hospitalization (days)	8.1 ± 1.2	5.9 ± 1.0

Note. Values reproduce the original M ± m notation; the dispersion measure is undefined and original p values could not be independently checked.

3.3 Intestinal culture findings

Reduced detection or abundance of Bifidobacterium spp. and Lactobacillus spp., greater opportunistic microbial growth and a higher frequency of grade III dysbiosis were recorded in the overweight/obesity group (Table 3). Associations were most pronounced for Bifidobacterium spp. and Lactobacillus spp., although a clinical definition of an abnormal culture and laboratory reference values were not provided. Culture techniques detect only selected organisms under specified cultivation conditions, limiting direct comparison with culture-independent profiling studies (Fan and Pedersen, 2021; Özçam and Lynch, 2024).

Table 3. Intestinal culture-based disturbances by weight group

Culture-defined disturbance	Overweight/obese (n = 30)	Normal weight (n = 30)	Fisher p
Reduced Bifidobacterium spp.	23 (76.7%)	9 (30.0%)	0.001
Reduced Lactobacillus spp.	20 (66.7%)	8 (26.7%)	0.004
Opportunistic microbial overgrowth	18 (60.0%)	7 (23.3%)	0.008

Culture-defined disturbance	Overweight/obese (n = 30)	Normal weight (n = 30)	Fisher p
Grade III dysbiosis	14 (46.7%)	4 (13.3%)	0.010

Note. Two-sided Fisher exact p values recomputed from source counts. Dysbiosis grading and microbiological thresholds require confirmation.

3.4 Upper-respiratory culture findings

Upper-respiratory Streptococcus pneumoniae, Haemophilus influenzae and Staphylococcus aureus detection were numerically more frequent among overweight/obese children (Table 4), but the count-based two-sided Fisher exact comparisons for individual taxa were not statistically significant. Mixed microbial culture was more common (53.3% vs 23.3%; Fisher p = 0.033). Because upper-airway colonization can occur in the absence of etiological involvement, these organisms should not be described as confirmed causes of the radiographic pneumonia without pathogen-specific supporting tests.

Table 4. Selected upper-airway culture findings by weight group

Detected organism/pattern	Overweight/obese (n = 30)	Normal weight (n = 30)	Fisher p
Streptococcus pneumoniae	13 (43.3%)	8 (26.7%)	0.279
Haemophilus influenzae	9 (30.0%)	4 (13.3%)	0.209
Staphylococcus aureus	7 (23.3%)	3 (10.0%)	0.299
Mixed microbial growth	16 (53.3%)	7 (23.3%)	0.033

Note. Two-sided Fisher exact p values calculated from source counts. Organism-specific categories may overlap; causality of pneumonia is not established by upper-airway sampling.

3.5 Reported changes during treatment

The original manuscript reported declining CRP and leukocytes following comprehensive therapy in the overweight/obesity group (Table 5). However, the definitions of the two columns used for cough and fever duration are unclear: duration is ordinarily assessed retrospectively after an episode, and the source simultaneously states that children were admitted within 72 hours of disease onset. The apparent inconsistencies require clarification against the case records. Because there was no adequately specified contemporaneous intervention control arm, no estimate of a microbiota-correction treatment effect can be inferred.

Table 5. Within-group outcomes reproduced from the original treatment table

Reported outcome	Before treatment	After treatment
C-reactive protein (mg/L)	32.4 ± 6.9	9.8 ± 2.7
Leukocytes ($\times 10^9$/L)	13.2 ± 1.9	8.9 ± 1.3
Cough duration (days)	12.1 ± 2.4	6.3 ± 1.5
Fever duration (days)	5.0 ± 0.9	1.8 ± 0.6

Note. The source-report wording and values are preserved. Time-point definitions, paired nature of the data, duration interpretation and source-reported $p < 0.001$ require verification before submission.

3.6 Correlations and biological interpretation

The original analysis reported a negative correlation between intestinal Bifidobacterium spp. counts and serum CRP ($r = -0.61$; source-reported $p < 0.01$) and a positive correlation between dysbiosis grade and hospital length of stay ($r = 0.58$; source-reported $p < 0.01$). The correlation type, analytic population, scale of microbial counts, potential confounders and underlying

data were not supplied. These associations are biologically plausible in view of gut–lung immune cross-talk (Dang and Marsland, 2019; Zheng et al., 2020), but they cannot demonstrate that intestinal microbial changes caused more severe pneumonia.

Host nutritional status may be associated with metabolic immune activation, epithelial barrier functions and microbial ecology. Such interactions could contribute to the clinical patterns observed here. Nevertheless, reverse causation is possible: acute infection, reduced food intake, antibiotic exposure and hospitalization may themselves alter culture-based microbial findings. Interpretation must therefore separate plausible biological pathways from mechanisms actually tested by the study (Özçam and Lynch, 2024).

Evidence supporting probiotic interventions in preventing upper-respiratory infections cannot be directly extrapolated to efficacy in treating established, radiologically confirmed childhood CAP. In particular, the intervention, dosing, initiation time and adverse-event outcomes in the present dataset are not described. The Cochrane review of preventive probiotic use addresses a different clinical question and illustrates the importance of intervention-specific evidence (Zhao et al., 2022).

3.7 Strengths and limitations

The reported study combined pediatric anthropometry, clinical outcomes, inflammatory measurements and paired-site culture assessments and included a weight-defined comparison group. Its principal limitations are small group sizes, incomplete baseline descriptors, absence of detailed weight-classification criteria, undefined bacterial abundance thresholds, use of selected bacterial cultures rather than comprehensive microbiome sequencing, absence of control-group results, no multivariable adjustment, uncertain continuous-variable dispersion, unverifiable original p values and missing patient-level data. The intervention protocol and duration-based endpoints are insufficiently described. These limitations preclude causal inference and strong treatment claims.

4. CONCLUSIONS

Among children aged 3–7 years hospitalized with community-acquired pneumonia, excess body weight was associated with higher observed frequencies of selected clinical symptoms, changes in culture-defined intestinal and upper-airway microbial findings, and higher source-reported inflammatory marker values. Exploratory analyses of aggregated counts supported some, but not all, of the original categorical comparisons. The findings are compatible with—but do not establish—the proposed involvement of gut–lung immune interactions in CAP severity. Studies with transparent age-standardized obesity definitions, clearly specified microbial methods, covariate-adjusted analyses and controlled, reproducible microbiota interventions are needed before a treatment recommendation can be justified.

ACKNOWLEDGEMENTS

The authors thank the clinical and laboratory staff of the Termez Branch of the Tashkent Medical Academy and the Republican Specialized Scientific-Practical Medical Center of Pediatrics for their assistance with patient recruitment, specimen collection and laboratory processing. The authors are grateful to the children and parents/legal guardians who agreed to participate in this study.

FUNDING

This study received no external funding. All costs associated with the study, including laboratory testing, data collection and manuscript preparation, were covered by the authors themselves.

AUTHOR CONTRIBUTIONS

Ch. L. Davronova — conceptualization, study design, clinical data collection, data analysis, drafting of the manuscript.

N.D. Azizova - patient recruitment, clinical assessment, laboratory data collection, critical revision of the manuscript.

F. M. Shamsiev — microbiological investigations, data curation, interpretation of results, critical revision of the manuscript.

Sh. B. Uzakova — supervision, methodology, statistical analysis, final approval of the manuscript.

All authors have read and approved the final version of the manuscript and agree to be accountable for all aspects of the work.

CONFLICT OF INTEREST

The authors declare no competing interests.

ETHICS APPROVAL AND INFORMED CONSENT

The study was conducted in accordance with the ethical principles of the Declaration of Helsinki and applicable national regulations. The study protocol was reviewed and approved by the [name of Ethics Committee. Written informed consent was obtained from the parents or legal guardians of all participating children, and assent was obtained from children where appropriate, after a full explanation of the nature and purpose of the study.

DATA AVAILABILITY

The anonymized datasets generated and analysed during the current study, including the aggregated clinical, laboratory and culture-based microbiological findings, are available from the corresponding author (Ch. L. Davronova, ch.davronova@yahoo.com) on reasonable request, subject to institutional data-sharing policies and ethical restrictions.

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