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MUCOCILIARY DYSFUNCTION IN RESPIRATORY PATHOLOGY: THE ROLE OF CILIARY EPITHELIAL PHASE SYNCHRONIZATION AND THE PERSPECTIVE OF MICROBIOME-ORIENTED CONTROL OF PATHOGEN COLONIZATION

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Introduction. Mucociliary clearance (MCC) is an integral mechanism of airway clearance, the effectiveness of which depends not only on ciliary beat frequency (CBF) but also on phase coordination and the metachronal organization of the ciliated epithelium. The CilioCode algorithm complements CBF assessment by analyzing phase coherence (phase-locking value, PLV), spectral entropy, and the network organization of ciliary motion (small-world index σ). Impairment of spatiotemporal coordination may reduce the efficiency of mucociliary transport and alter the conditions of interaction between microorganisms and the epithelial surface. Neuroendocrine cells (NECs) are regarded as potential sensory components of neuroimmune regulation of the airways; however, their presence and functional significance specifically in the human nasal mucosa remain insufficiently defined. The spectrum of respiratory pathogens and their antimicrobial susceptibility is also of clinical importance: the detection of resistant strains supports the search for alternative methods of pathogen control, including our proposed use of airway commensals modified using the clustered regularly interspaced short palindromic repeats (CRISPR) system (*Clustered Regularly Interspaced Short Palindromic Repeats*).

Objective. To characterize changes in mucociliary clearance (MCC) in viral, bacterial, and obstructive profiles of respiratory pathology; to determine the phase coordination of the ciliated epithelium and assess its association with functional transport; to characterize the spectrum of pathogens and the resistance profile in the bacterial phenotype as a basis for microbiome-oriented approaches that could potentially be used in the treatment of viral and fungal

pathologies of the upper and lower respiratory tracts.

Materials And Methods. A prospective study was conducted at the Municipal Non-Profit Enterprise “City Children's Polyclinic No. 15” of the Kharkiv City Council, Non-Profit Enterprise “City Children’s Clinical Hospital No. 16” of Kharkiv City Council, “City Polyclinic No. 24” of Kharkiv City Council, and Kharkiv National University of Radio Electronics. A total of 354 individuals were examined: a control group (n = 45) and 309 patients distributed into three mutually exclusive groups according to the predominant phenotype: A (viral phenotype, n = 136) — a positive reverse transcription polymerase chain reaction (RT-PCR) test in the absence of microbiological verification of a bacterial pathogen and with a procalcitonin (PCT) level <0.25 ng/mL; B (bacterial phenotype, n = 60) — a microbiologically verified bacterial pathogen and PCT ≥0.5 ng/mL; cases that did not meet these criteria or had signs of a mixed phenotype were not included in the comparative analysis; C (non-febrile nasal obstruction, n = 113) comprising three subtypes: prolonged severe (3–5 years), partial (≤6 months), and acute severe (≤1 month).

In groups A and B, lung ultrasound (LUS) (GE Vscan Air CL) was performed to assess the lung parenchyma and exclude sonographic signs of consolidation. All patients were assessed using the NeuroGlio-Capillary System Risk Score (NGCS-RS). Pathogens were verified only in group B: bacterial culture with identification by matrix-assisted laser desorption/ionization time-of-flight mass spectrometry (MALDI-TOF); antimicrobial susceptibility was determined according to the current criteria of the European Committee on Antimicrobial Susceptibility Testing (EUCAST), version 16.1. Culture-isolated pathogens and atypical pathogens were detected by polymerase chain reaction (PCR).

In all groups, *in vivo* mucociliary clearance (MCC) was assessed by the saccharin transit test (STT), and nasal patency was assessed by rhinomanometry. Following the STT, a brush sample of ciliated epithelium was obtained from the inferior nasal turbinate, followed by video microscopy in Dulbecco’s phosphate-buffered saline (DPBS) at 37 ± 0.5 °C, 128 frames/s, a resolution of 640 × 480, and a 100× objective; ciliary beat frequency (CBF), phase

synchronization quantified by the phase-locking value (PLV) using the Hilbert transform, spectral entropy (Morlet wavelet), and the small-world index σ (σ > 1 — *small-world* topology; 1000 null networks) were determined. PLV is a measure of phase synchronization between signals that can be calculated on the basis of instantaneous phases obtained using the Hilbert transform.

Statistical analysis: normality of the distribution was assessed using the Shapiro–Wilk test; between-group differences were assessed by one-way analysis of variance (ANOVA), and pairwise comparisons were performed with Bonferroni correction; correlations were assessed using partial Spearman correlation adjusted for age and sex; associations with STT were assessed using multiple linear regression; discriminative ability was assessed using receiver operating characteristic analysis (ROC analysis), with comparison of the areas under the curves (AUCs) by the DeLong method. The statistical significance level was $p < 0.05$. For regression models, β-coefficients, 95% confidence intervals (CIs), and the adjusted coefficient of determination (adjusted R^2) were reported.

Results. In group A, peripheral blood oxygen saturation (SpO_2) was 96.8 ± 1.2%, NGCS-RS was 3.2 ± 1.6 points, and C-reactive protein (CRP) was 4.1 ± 1.8 mg/L; no sonographic signs of parenchymal consolidation were detected by LUS. In group B, SpO_2 was 91.2 ± 2.8%, NGCS-RS was 11.8 ± 2.6 points, and CRP was 42.7 ± 28.3 mg/L; no consolidation was detected by LUS either. Among the pathogens (with the proportion of resistant isolates shown in parentheses), *Streptococcus pneumoniae* (*S. pneumoniae*) was detected in 36.6% (azithromycin 31%, ceftriaxone 0%), *Mycoplasma pneumoniae* (*M. pneumoniae*) in 23.2% (intrinsic nonsusceptibility to β-lactams, azithromycin 14%), *Haemophilus influenzae* (*H. influenzae*) in 16.4% (ampicillin 10%, amoxicillin/clavulanic acid 0%), *Bordetella pertussis* (*B. pertussis*) in 10.0% (macrolides 0%), *Klebsiella pneumoniae* (*K. pneumoniae*) in 6.7%, and *Moraxella catarrhalis* (*M. catarrhalis*) in 6.7% (β-lactamase production 100%). In group C, SpO_2 was 94.5 ± 1.9%, NGCS-RS was 6.1 ± 2.2 points, CRP was 8.6 ± 5.1 mg/L, and PCT was 0.08 ± 0.06 ng/mL. The values of phase-locking value (PLV), small-world index (σ), ciliary beat frequency (CBF, Hz), spectral entropy, and the saccharin transit test

(STT, min) in the control group were 0.86 ± 0.05; 2.4 ± 0.3; 12.1 ± 1.2; 11.3 ± 2.1, respectively. In group A, they were 0.69 ± 0.13; 1.8 ± 0.4; 10.5 ± 3.0; 15.5 ± 7.0, and in group B — 0.58 ± 0.13; 1.4 ± 0.4; 9.0 ± 3.5; 28.0 ± 7.0, respectively. In group C, in prolonged severe obstruction they were 0.42 ± 0.06; 1.1 ± 0.2; 7.3 ± 1.4; 24.5 ± 3.1, in partial obstruction — 0.63 ± 0.08; 1.5 ± 0.2; 7.6 ± 3.1; 18.0 ± 2.4, and in acute severe obstruction — 0.40 ± 0.05; 1.0 ± 0.2; 6.8 ± 2.2; 34.7 ± 3.3. The groups differed in PLV ($F = 138.6$), σ (109.4), spectral entropy (127.8), STT (118.3), and CBF (24.1), all $p < 0.001$. Prolonged and acute severe obstruction were characterized by dyssynchrony, whereas partial obstruction showed only partially preserved coordination. The σ index exceeded 1 in the control group and in partial obstruction, approached the threshold value in prolonged severe obstruction ($\sigma = 1.1$), and corresponded to the threshold value in acute severe obstruction ($\sigma = 1.0$). Prolonged and acute severe obstruction did not differ in PLV ($p = 0.38$) or σ ($p = 0.42$), but had different STT values; this may indicate an additional, hypothetically rheological component of transport that requires direct rheometry of mucus. PLV correlated with STT ($\rho = -0.78$) more strongly than CBF ($|\rho| = 0.32$), whereas spectral entropy showed a correlation of $\rho = 0.71$. In the internal regression analysis, the model including PLV and spectral entropy explained 71% of the variability in STT ($R^2 = 0.71$), whereas the model including CBF explained 12% ($R^2 = 0.12$). According to ROC analysis, the AUC for PLV was 0.91, for CBF 0.67, and for the integrated model 0.96 (95% CI 0.91–0.99; internal assessment only, without external validation).

The resistance profile (32% azithromycin-resistant *S. pneumoniae*; intrinsic nonsusceptibility of *M. pneumoniae* and *K. pneumoniae* to β-lactams) limits the options for antibiotic therapy. A hypothetical alternative is CRISPR-modified nasal commensals *Corynebacterium accolens* and *Dolosigranulum pigrum*, whose activity may be based on niche competition, antimicrobial metabolites, and controlled local secretion of hIL-10 (modulation of NF-κB/STAT3); biosafety should be ensured by a kill-switch. This strategy primarily concerns nasopharyngeal colonizers — *S. pneumoniae*, *H. influenzae*, and *M. catarrhalis* (36 of 60 isolates), whereas atypical and intracellular pathogens will likely remain outside

its scope of action. Its efficacy and safety should be evaluated in preclinical models, in which PLV and σ may serve as functional indicators of epithelial status; the strategy was developed by authors, but was not tested in the present study.

Conclusions. Impairment of mucociliary clearance (MCC) in respiratory phenotypes was accompanied not only by a reduction in ciliary beat frequency but also by impaired phase coordination and network organization of the ciliated epithelium. The phase-locking value (PLV) demonstrated a stronger association with the saccharin transit test (STT) than ciliary beat frequency (CBF), while integration of PLV and spectral entropy showed higher internal discriminative ability. In the bacterial phenotype, a clinically relevant spectrum of respiratory pathogens and resistance was identified. The results support further investigation of microbiome-oriented colonization-resistance strategies; however, their efficacy and safety require separate preclinical validation.

CURRENT APPROACHES TO THE PHARMACOTHERAPY OF INFECTIONS CAUSED BY CARBAPENEM-RESISTANT *ACINETOBACTER BAUMANNII*

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Introduction. Infections caused by carbapenem-resistant *Acinetobacter baumannii* (CRAB) pose a major therapeutic challenge due to the limited range of active antibiotics and the toxicity of certain treatment regimens. In Ukraine, the significance of this problem is further heightened by the prevalence of resistant isolates among pathogens recovered from combat wounds. The introduction of sulbactam–durlobactam and cefiderocol has expanded the available therapeutic options; however, differences in their mechanisms of action and clinical evidence base require a differentiated assessment. Particular importance should be placed on distinguishing in vitro antimicrobial activity from clinically confirmed efficacy.

Aim. To analyse the pharmacological rationale, clinical efficacy, and safety of sulbactam–durlobactam, cefiderocol, and combination

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