Background and Aims Second disbioses of the respiratory play the presentative role and had negatively influence on the result of duration of infectious diseases and assists development of immune dysfunction in mucous membranes of respiratory tract.

Methods We investigated 24 children with the first Diagnosed Pulmonary Tuberculosis (FDPT) in the age from 1–16 years. Research of microflora of respiratory tracts was conducted by a bacteriologic examination of native material (expectoration).

Results The inspected contingent had Pulmonary form of the first diagnosed tuberculosis. The patients concluded: child to 3 years - 50.00%. other 50.00% children contained the group of puberat period. Distributed on the forms of tubercular process: primary tubercular complex - 25.00%, pulmonary focus tuberculose 12.50%, disseminated tuberculosis - 25.00%, infiltrative tuberculosis - 37.50%. 58.30% children had assoctation pathologi with the FDPT: anaemia in 25.00% cases, pneumonia - 8.30%, HIV - 8.30%. In microbiological culture was confirmed presence of M. tuberculosis in 33.30% cases. The destructive chang in lung 16.70% cases was identified. N.sicca was present in 40.00% children with the normal microflora and S.epidermidis - in 60.00%. In 50.00% cases of children with the FDPT disbioses violations was identified after the beginning of using of antiphthtic treatment. Disbiosis as a monoculure found in 83.30% cases, in 16.70% cases - as associations of cultures. In 33.30% cases found out Escherichia coli, in the 16.70% - K.pneumonia, in the 50.00% cases - Candida A.

Conclusions On the basis of the conducted researches are set presence of respiratory disbiosis in children with the FDPT.

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A NEONATAL RAT MODEL OF BRONCHOPULMONARY DYSPLASIA INDUCED BY PRE- AND POSTNATAL INFLAMMATION WITHOUT EXPOSURE TO HYPOXIA

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Purpose We tested if pre- or postnatal inflammation can develop BPD per se and if there are any interaction between prenatal and postnatal inflammation.

Methods Two days before delivery (E20), 1 µg of lipopolysaccharide (LPS) or vehicle (V) was injected into each amniotic sac, and after birth 0.25 mg/kg of LPS or vehicle was injected into peritoneum on P1, F3, and P5. This led to four experimental groups. On P7 and P14, their lungs and hearts were harvested, and alveolarization and lung vascular density were evaluated.

Results Morphometric analysis of P7 lungs revealed that both preLPS+postLPS group and V+postLPS group had significantly larger and less complex airspaces and small alveolar surface area than V+V group. On P14, only postLPS group had significantly larger and less complex airspaces than V+V group. However, alveolar surface areas were significantly smaller both in preLPS+postLPS group and V+postLPS group than in V+V group. Lung vascular density of both preLPS+postLPS group and V+postLPS group was significantly lower than V+V group.

Conclusions At these intra-ammiotic and postnatal systemic LPS doses, prenatal intra-ammiotic LPS injection per se did not affect postnatal alveolar and pulmonary vascular development, while postnatal systemic LPS injection significantly inhibited alveolar and pulmonary vascular development regardless of whether prenatal intra-ammiotic LPS was injected or not. There was no definite interaction between intra-ammiotic LPS and postnatal systemic LPS on the lung development. This rat model of BPD could be used as a valuable tool for testing the effect of anti-inflammatory agents on the prevention of BPD.